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SACRO-ILIAC ARTHRITIS

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SEVERAL years ago, in discussing age changes as found in macerated skeletons, the writer made the statement that the sacro-iliacs were ankylosed by bone productive changes more frequently than any other joints, and that they were often completely fixed when the other parts of the same skeleton showed little if any similar change.

A recent review of 1559 human skeletons in the Hamann Museum of Anthropology disclosed 96 in which one or both sacro-iliac joints are firmly ankylosed by calcification of ligaments and bone lipping. The material of this museum is collected through the dissecting room, the city morgue, and various city and county institutions. It consists mostly of those individuals of various races who have failed to make a place for themselves in the local social arrangement, and therefore probably presents a higher incidence of disease and defect than a true cross section of the local population.

In addition to the 96 specimens showing complete sacro-iliac ankylosis, a much greater number presented similar bone changes not of sufficient degree to resist disarticulation during maceration.

A most arresting feature of the osteogenic changes was the occurrence of two types developing independently. Sometimes a skeleton shows one type alone, but more often the two are co-existent. The one is a smooth calcification of the anterior sacro-iliac ligament; the other an irregular lipping at the joint periphery. The former is present in younger subjects, often involves other parts of the spinal column but not the skeleton in general, and is a true spondylitis of the axial skeleton. The latter is predominant in older individuals, a hypertrophic type, usually of general distribution through the entire skeleton. A few specimens were found which showed complete obliteration of the joint with no other pathology apparent. These were considered congenital synostoses.

Spondylitis of the ankylosing type has been discussed under various titles, such as spondylitis deformans, spondylose rhizómelique, spondylitis atrophica ligamentosa, Bechterew's and Marie-Strumpel's disease; each differing in such minor factors as to joints most involved, the presence of nerve-root irritation, muscle weakness, meningitis, and the ultimate posture of fixation; all, however, having the major characteristics of bone atrophy, synovial proliferation, pannus formation, calcification of ligaments, and final fixation.

Ankylosis of sacro-iliac joints due to this type of disease was found in 19 pelves. The calcification, which because of its physical appearance has been likened to candle drip, was found most frequently at that part of the joint in the pelvic brim, extending through the anterior ligament to form a bridge of bone

about 1/8 inch thick spanning the joint line. In its earlier stages the process is seen extending from both sacrum and ilium toward a common center. Posterior to the joint there is no capsular ligament. Here the strong irregular fibrous bands anchoring ilium to sacrum are rarely calcified. The average age of these 19 individuals was 45.9 years.

In 6 subjects ankylosis was due to lesions of distinctly hypertrophic type, irregular exostoses fusing or interlocking across the joint. These averaged 67 years of age. The almost universal appearance of joint lipping at about the fortieth year of life and its progress with age must be considered here. Since joint structures respond in similar manner to chronic irritation by different agents, it is impossible to estimate the relative importance of several associated factors in the production of given pathological changes. Practically all of the individuals that make up this material present at least the three factors of age, mechanical strain, and focal infection; each tending to produce circulatory changes with disturbance of calcium metabolism. The writer admits his present inability properly to evaluate the various factors of the problem.

In 67 skeletons the two types of pathology were co-existent in various degrees of severity. These subjects averaged 57.3 years of age and all showed general joint lipping. Some of the older individuals particularly showed smooth calcification of the anterior sacro-iliac ligament and marked hypertrophic exostoses of their lumbar vertebræ. The co-existence of the two types of arthritis in so great a number of skeletons suggests that they are of similar or closely related etiology. This coincides with the beliefs of H. Warren Crowe, Reginald Burbank, and others, who from their exhaustive researches attribute the different types of arthritis to Streptococcus, hæmolyticus and viridens, and staphylococcus infections. Though they differ as to which is which, both insist that the great majority of arthritides are due to the combined activity of these organisms, and both present strong therapeutic evidence to substantiate their claims.

Of the four pelves presenting obliteration of a sacro-iliac joint, probably congenital, the left side was involved in 3; in 1, the right. One was a white female 72 years old, 2 were colored women, 36 and 33 years old, respectively. The other was a negro male of 31 years. The preponderance of females in this very small group is much the reverse of the series as a whole. Only 2 of the 92 with arthritic ankylosis were of this sex.

Embryologically the ilium develops in the hind limb bud and approaches the axial skeleton, making contact with the first, second, and third sacral segments in the seventh week of fetal life to form the sacro-iliac joint. This articulation thus differs fundamentally from those formed in the usual manner by vacuolation of the skeletal blastema between areas of chondrification. Ordinarily it presents all the features of a typical joint; synovia, articular cartilages, and ligaments; but the synovial cavity is relatively small, the cartilages are

Fig. 1. Photograph and roentgenogram of congenital obliteration of a sacro-iliac joint.

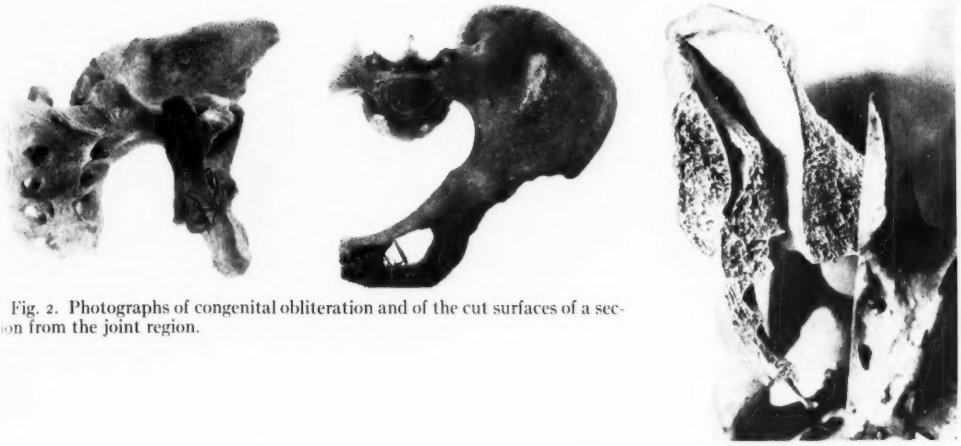

Fig. 2. Photographs of congenital obliteration and of the cut surfaces of a section from the joint region.

thin, their apposing surfaces irregular, the ligament incomplete posteriorly, and mobility very slight. Considering these facts one is rather surprised at the incidence of only four synostoses in 1559 skeletons.

To determine the deeper structure of a synostosis and the possibility of its being only a superficial fusion, a section of bone was removed from the back of specimen 1750. This wedge cut through the joint area in two directions. The cut surface showed condensation of bone trabeculæ in the area where the joint line should have been, but no interruption of bone architecture. If there had ever been a joint present all trace of its existence was lost. Shore has recorded similar findings in such an anomaly. Though others have written of sacro-iliac obliteration their descriptions are most suggestive of ankylosis.

The function of the hind limb is propulsive power. The first requirement of its articulation with the axial skeleton is stability. Since the assumption of the upright posture accentuates the need of stability we find that in man the sacro-iliac joint is relatively larger in all dimensions than in other animals; including

Fig. 3. The calcified structure has been broken, showing its thin superficial character.

Fig. 4. With and without the sacrum. Smooth calcification of the right, irregular lipping of the left sacro-iliac.

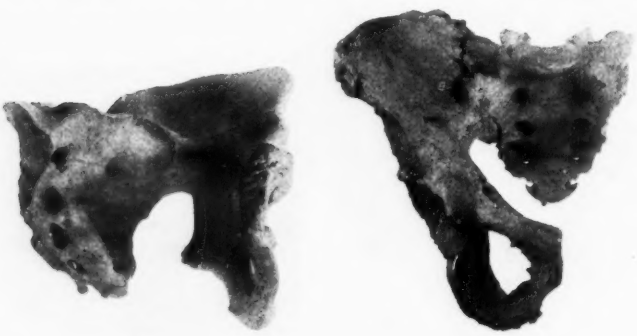

Fig. 5 Fig. 6

Fig. 5, left. Smooth superficial calcification of the left sacro-iliac, complete except at the brim. This reverses the usual distribution. Fig. 6. Irregular lipping of hypertrophic type.

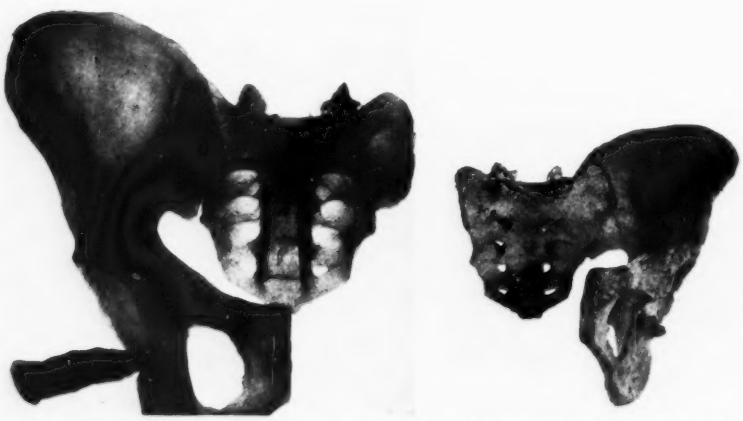

Fig. 7. Roentgenogram and photograph, contrasting evidence shown by transmitted and reflected light.

more segments of the sacrum and presenting greater surfaces for attachment of ligaments (see Straus). Ankylosis is nature's method of stabilizing an insecure joint.

Though a clinical discussion of sacro-ili arthritis will be endeavored in a later pap it is perhaps permissible here to compare t evidence of ankylosis as shown by X-ray a

Fig. 8. Posterior ankylosis without narrowing of the joint line. Calcification of the posterior sacro-iliac ligament is exceptional. It is demonstrated by dorsovertical roentgenogram.

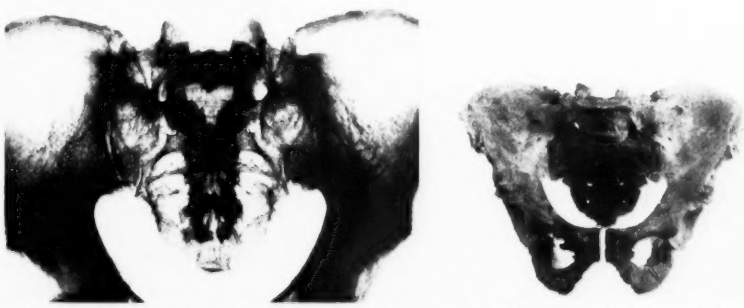

Fig. 9. Note the difference in X-ray and photographic evidence of calcification and ankylosis. The sacrum is fixed by fusion of the last lumbar vertebra with both ilia.

photograph. Such a comparison compels one to the opinion that the points of importance in interpreting the ordinary X-ray film are not the presence or absence of lipping at the upper and lower poles of the joint. These are the sites least affected. Neither is narrowing of the joint line dependable. There may be complete ankylosis without loss of cartilage thickness. The features of most significance are change in bone density and loss of clarity of joint outline, particularly at the pelvic brim. When actual lipping or calcification has occurred (and, of course, arthritis has existed for some time by then), ventro-or dorso-vertical X-ray views may be required to demonstrate it. The former is used by obstetricians in roentgenographic pelvimetry; the latter, to demonstrate abnormalities of the sacral canal.

The material from which this study was made comprised 1559 skeletons, 392 of which

TABLE I.—A DETAILED CHART OF 96 SPECIMENS OF SACRO-ILIAC ANKYLOSIS FOUND IN 1559 SKELETONS

Type	Cad. No.	Color	Sex	Age	Side			Site				Lipping	
					Right	Left	Both	Front	Back	Upper	Lower	Axial	Acet.
Congenital	1266	N	F	33		L						+	+
	1750	W	F	72		L						+	+
	1871	N	F	36	R								
	1876	N	M	31		L							
Total	4	3 1	3 1	Av. 43	1	3	0					2	2
Spondylitic	458	N	M	30			B	+		+			+
	475	N	M	50		L				+		+	+
	1722	N	M	39	R								
	317	W	M	27	R			+			+		
	549	W	M	32	R			+					
	602	W	M	35		L		+		+	+	+	+
	459	W	M	40		L		+		+			
	1680	W	M	42			B	+					
	1237	W	M	49		L				+		+	
	1076	W	M	44		L	B	+		+			+
	465	W	M	50	R			+				+	+
	102	W	M	50		L		+		+		+	+
	1404	W	M	53			B	+	+	+	+	+	+
	1023	W	M	52	R			+				+	+
	674	W	M	50			B	+					+
	206	W	M	62			B	+		+		+	+
	299	W	M	65	R			+	+	+	+	+	+
	1259	W	M	78		L		+				+	+
	923	N	F	23		L		+					
Total	19	4 15	1 18	Av. 45.9	6	7	6	16	2	9	4	10	12
Hypertrophic	1045	W	M	40			B					+	
	186	W	M	68	R				+		+	+	+
	1728	W	M	70			B	+		+	+	+	+
	1519	W	M	71	R			+		+		+	+
	399	N	M	70	R			+	+			+	+
	1335	W	M	75			B	+	+	+	+	+	+
Total	6	1 5	6	Av. 67+	3		3	4	3	3	3	6	6
Mixed spondylitic and hypertrophic	1666	N	M	43	R			+		+			+
	198	N	M	48			B	+		+		+	+
	1416	N	M	48			B	+		+		+	+
	1470	N	M	51	R			+				+	+
	1377	N	M	55			B	+		+		+	+
	1408	N	M	65			B	+	+			+	+
	1528	N	M	67			B	+		+		+	+
	1655	N	M	80			B	+		+		+	+
	1340	N	M	88			B	+		+		+	+
	1857	N	M	61			B	+		+		+	+

TABLE I.—A DETAILED CHART OF 96 SPECIMENS OF SACRO-ILIAC ANKYLOSIS FOUND IN 1559 SKELETONS—Continued

Type	Cad. No.	Color	Sex	Age	Side			Site				Lipping	
					Right	Left	Both	Front	Back	Upper	Lower	Axial	Acet.
	1433	W	F	81			B	+	+		+	+	+
	1593	W	M	45	R			+		+	+	+	+
	444	W	M	45			B			+			+
	285	W	M	45		L		+		+		+	+
	1007	W	M	45			B	+		+		+	+
	1118	W	M	45		L			+			+	+
	666	W	M	47			B	+		+		+	+
	1001	W	M	49	R			+		+		+	+
	1645	W	M	50			B	+		+		+	+
	382	W	M	50	R					+		+	+
	200	W	M	50		L				+		+	+
	239	W	M	50		L		+		+		+	+
	509	W	M	52			B	+		+		+	+
	1181	W	M	52	R					+	+	+	+
	372	W	M	52			B	+		+		+	+
	1629	W	M	53		L		+		+		+	+
	300	W	M	55	R			+		+		+	+
	1455	W	M	50			B	+		+		+	+
	789	W	M	54	R			+				+	+
	418	W	M	54	R			+				+	+
	1428	W	M	54	R			+		+		+	+
	1566	W	M	54			B	+				+	+
Mixed spondylitic and hypertrophic	1598	W	M	55			B	+		+		+	+
	659	W	M	55			B	+				+	+
	776	W	M	55		L		+	+			+	+
	797	W	M	57		L		+				+	+
	1019	W	M	58		L		+				+	+
	1636	W	M	58			B	+				+	+
	663	W	M	60		L		+		+		+	+
	1464	W	M	60		L		+		+		+	+
	1198	W	M	60		L		+		+		+	+
	884	W	M	60	R			+				+	+
	19	W	M	60			B	+				+	+
	167	W	M	60			B	+	+	+	+	+	+
	1660	W	M	64		L		+				+	+
	349	W	M	63		L		+	+			+	+
	363	W	M	64		L		+				+	+
	1113	W	M	65			B	+		+		+	+
	1218	W	M	65	R			+				+	+
	1640	W	M	67			B	+	+	+	+	+	+
	309	W	M	66		L		+		+		+	+
	1780	W	M	66			B	+		+		+	+

TABLE I.—A DETAILED CHART OF 96 SPECIMENS OF SACRO-ILIAC ANKYLOSIS FOUND IN 1559 SKELETONS—Continued

Type	Cad. No.	Color	Sex	Age	Side			Site				Lipping	
					Right	Left	Both	Front	Back	Upper	Lower	Axial	Acet.
	353	W	M	68			B	+		+		+	+
	1303	W	M	69	R			+				+	+
	1396	W	M	70			B	+		+		+	+
	304	W	M	70	R			+		+		+	+
	1490	W	M	72			B	+		+		+	+
	1239	W	M	74	R			+				+	+
	1260	W	M	77			B	+				+	+
Mixed spondy-iltic and hypertrophic	1017	W	M	77			B	+		+		+	+
	1578	W	M	79			B	+		+		+	+
	1596	W	M	79			B	+		+		+	+
	397	W	M	81			B	+		+		+	+
	115	W	M	88	R			+		+		+	+
	821	W	M	79		L			+			+	+
	1366	N	M	40			B			+		+	+
	1423	W	M	45		L		+		+		+	+
Total	67	11 56	1 66	Av. 57.2	16	17	34	59	7	45	6	66	67

TABLE II.—SYNOPSIS WITH MODES AND AVERAGES OF TABLE I

Type	No.	Color W — N	Sex M — F	Average age	Side			Site				Lipping	
					Right	Left	Both	Front	Back	Upper	Lower	Axial	Acet.
Congenital	4	1 3	1 3	43	1	3	0	4	4	4	4	2	2
Spondylitic	19	15 4	18 1	45.9	6	7	6	16	2	9	4	10	12
Hypertrophic	6	5 1	6 0	67	3	0	3	4	3	3	3	6	6
Mixed	67	56 11	66 1	57.2	16	17	34	59	7	45	6	66	66
Totals	96	77 19	91 5	55	26	27	43	79	16	61	17	84	86

were of colored males, 105 colored females, 125 white females, and 936 white males. It appears then that the incidence of sacro-iliac ankylosis is proportionately nearly twice as great in the negro as in the white subjects, and practically three times as frequent in the male as the female. There is no preference as to right and left side. It is nearly as often bilateral as unilateral. That part of the joint which takes part in the formation of the pelvic brim is by far the most frequently involved. The process extends from the brim upward much more than downward, and seldom involves the lower pole of the joint. In other words the bone and joint changes are in direct ratio to mechanical stresses of upright posture.

In individuals more than 40 years old lipping of the axial skeleton and acetabulum is practically universal, and increases with age

CONCLUSIONS

1. From the standpoint of skeletal change the sacro-iliac joint is more frequently and more extensively involved in arthritic lesions than any other joint of the body.

2. Two distinct, usually co-existent, types of arthritis affect the sacro-iliac joint.

3. Because of its manner of development the sacro-iliac is subject to embryonic defect and thus shows congenital obliteration more frequently than other joints.

4. Since the joint retains in man the ana

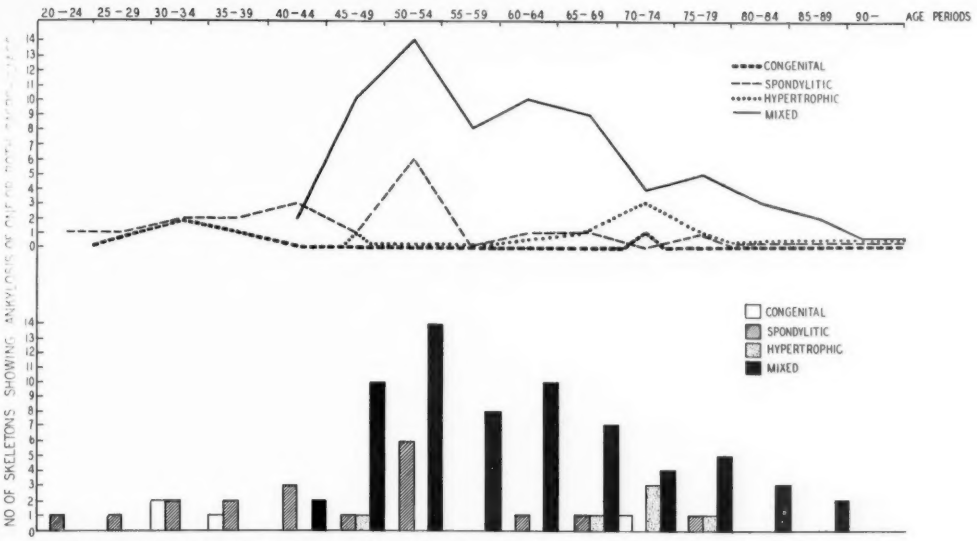

Fig. 10. Chart showing incidence of the different types of sacro-iliac ankylosis in relation to age factor.

tomical features of its quadripedal origin the mechanical stresses peculiar to the upright posture subject it to ligamentous strains. Ankylosis is a compensatory mechanism for stabilization.

5. Current roentgenographic criteria for determination of sacro-iliac joint defect are in urgent need of revision.

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INFRAPULMONARY EMPYEMA[1]

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INFRAPULMONARY ("diaphragmatic," "supradiaphragmatic") empyema is a type of encapsulated suppurative pleuritis in which the effusion is interposed between the lower lobe of the lung and the diaphragm. Its difficulty of recognition, the fact that often it is undiagnosed until the late stages, the frequency with which it may be confused with certain pulmonary and extrapulmonary conditions, and finally the technical problems which may arise at the time of operation, all contribute not only to its general interest but also to its importance.

In spite of its many interesting features apparently only scant attention has been paid to the subject in the past, and a search of the recent literature has failed to disclose a single reference to the subject by title. While the lesion is well known and has been described in textbooks (5) and reported in articles dealing with encapsulated empyema (10), it generally has been regarded merely as an unusual variety of the latter, diagnoseable as a rule only by X-ray examination or occasionally unexpectedly encountered at postmortem section. Under these circumstances, it is not surprising that comparatively little has been written concerning its clinical, diagnostic, or therapeutic aspects. In our experience, the lesion while not common has been by no means as rare as generally assumed and of late has come to be pre-operatively recognizable with increasing frequency.

This communication is based on observations made in a series of 12 cases[2] from the Surgical Service of Dr. Harold Neuhof at the Mt. Sinai Hospital, New York. Four additional cases were encountered in a search of the general files of the hospital. Inasmuch as the latter were not observed personally by the author, they have been utilized only to the extent of recording the type of organism present and the nature of the underlying causative pulmonary process. In this paper we will confine ourselves to a discussion of the practical aspects of the subject, based on the study of our material. Since a statistical review of such a small series is apt to be inconclusive, figures for the most part have been eliminated.

DEFINITION

"Infrapulmonary empyema" is a term originated by Wessler to designate an encapsulated collection of pus, situated between the under surface of the lower lobe of the lung and the diaphragm. The term "infrapulmonary" rather than "diaphragmatic" or "supradiaphragmatic" empyema is employed because it most accurately describes the lesion. For example, an encapsulated empyema situated in the lowermost part of the general pleural cavity may extend downward into the costophrenic sinus and make contact with the periphery of the diaphragm. This may be broadly classified by some as a supradiaphragmatic empyema, but the use of the more precise term "infrapulmonary empyema" immediately eliminates it from our consideration.

ETIOLOGY

1. As in other varieties of pleural suppuration, infrapulmonary empyema most commonly is the result of extension of infection from the lung into the adjacent pleura. In this connection it should be stated that it may exist as the sole pleural lesion, or as a secondary part of a more widespread pleural suppurative process.

2. A second cause is the extension of infection from beneath the diaphragm, as in subphrenic or liver abscess.

This discussion is specifically limited to the group in which the infrapulmonary empyema is derived from infection in the lung and represents the sole pleural lesion. All of the cases in this series fall precisely into the above group.

BACTERIOLOGY

As is the case in other varieties of pleural suppuration, no specific type of organism

[2] Since submitting the manuscript for publication, 3 additional cases of infrapulmonary empyema have been observed by the author.

[1] From the Surgical Service of Dr. Harold Neuhof, Mt. Sinai Hospital. Delivered before the Surgical Section of the New York Academy of Medicine, May 5, 1933.

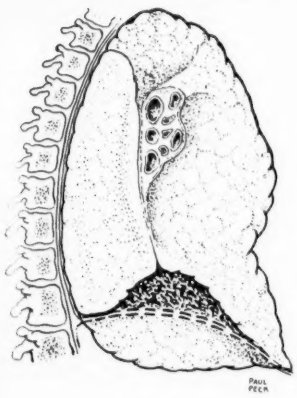

Fig. A.

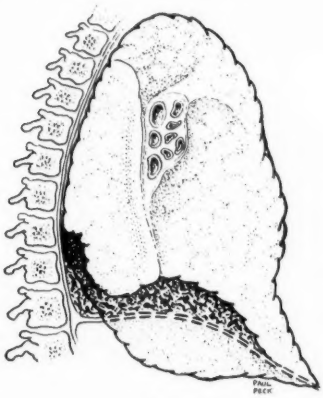

Fig. B.

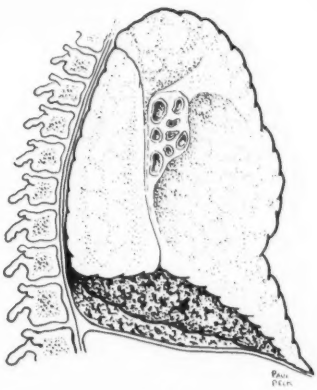

Fig. C.

Fig. A. Mediastinal aspect of the left lung. Group 1 lesion. Diaphragm indicated by broken lines. Collection of pus situated between dome of diaphragm and under surface of lung. Note that empyema is separated from the chest wall anteriorly, laterally, and posteriorly by the lappet of lower lobe which dips into the costophrenic sinus.

Fig. B. Mediastinal aspect of left lung. Group 2 lesion. Diaphragm indicated by broken lines. Collection of pus between dome of diaphragm and under surface of lung extends posteriorly, and there comes into contact with the chest wall after elevating the posterior margin of the lung. Collection then enlarges chiefly in the region behind, rather than beneath the lower lobe. Note that empyema is separated from the chest wall only on the anterior and lateral aspects by the lappet of lower lobe which dips into the costophrenic sinus.

Fig. C. Mediastinal aspect of left lung. Group 3 lesion. Diaphragm indicated by solid lines. Collection of pus between dome of diaphragm and under surface of lung extends laterally and posteriorly, and comes into contact with the chest wall after elevating the lateral and posterior margins of the lung. Collection remains loculated beneath the lower lobe, however, and does not extend into general pleural cavity as in Group 2. Note that the lappet of lower lobe anteriorly, still remains adherent in the costophrenic sinus.

responsible for infrapulmonary empyema, and the organism present varies according to the type existing in the underlying pulmonary source of infection. The following types of bacteria, in their order of frequency, were noted: pneumococcus, 9 cases; mixed anaerobes, 3 cases (3); Staphylococcus aureus, 1 case; and negative smear and sterile culture, 3 cases.

PATHOLOGY AND PATHOGENESIS

The essential pathological feature is the existence of an infective lesion involving the basilar portion of the lower lobe, with the subsequent development of a suppurative process in the subjacent pleural space between the under surface of the lower lobe and the diaphragm. The following pulmonary lesions, in the order of their frequency, have been noted in this series: lobar or bronchopneumonia, 12 cases; ruptured putrid lung abscess, 3 cases; and necrosis of an infected pulmonary infarct, 1 case.

For purposes of description and classification the cases have been divided into three groups:

1. The first group is comprised of cases in which the purulent collection remains localized between the lower lobe and the diaphragm, and does not come into contact with the chest wall at any point. In the early stage the lesion is small, and the portions of the under surface of the lobe peripheral to it become adherent to the surface of the diaphragm as a result of the existing inflammatory reaction. The purulent collection thus becomes encapsulated, and completely separated from the chest wall on all aspects. As the empyema enlarges, it gradually peels more and more of the under surface of the lobe away from the diaphragm, until finally the two are separated over a comparatively wide area. At the extreme periphery, however, the margin or lappet of the lower lobe remains firmly adherent to the diaphragm in the region of the costophrenic sinus. Although this por-

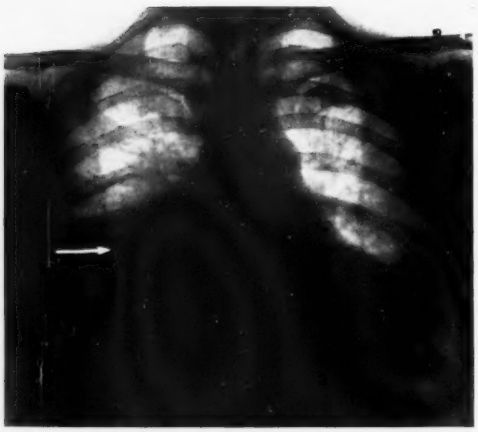

Fig. 1. C.D. Case 11. Infrapulmonary empyema. Early stage. Film reveals only consolidation (indicated by arrow) of the right lower lobe, diagnosed as pneumonia. Small empyema beneath the lower lobe is obscured by the pulmonary infiltration. Group 1 lesion.

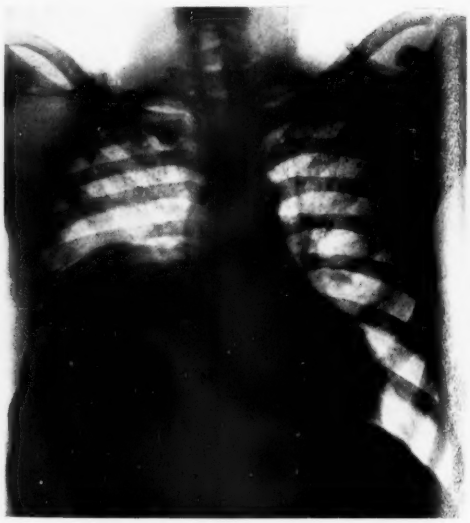

Fig. 2. L.D. Infrapulmonary empyema. Dense shadow with convex upper border in lower half of right chest. Moderate cardiac displacement. Lesion indistinguishable from elevated diaphragm. Group 3 lesion.

tion of the lung may eventually become considerably thinned out and compressed between the tense collection of pus and the chest wall, it nevertheless remains interposed between the two (Fig. A).

2. The second group is comprised of cases in which the purulent collection after reaching a variable size, dissects its way beneath the margin of the lower lobe at a certain point, and there comes into contact with the chest wall. This can occur only in an area in which the margin has failed to become, or remain, firmly adherent to the diaphragm. The usual site of such failure is posteriorly, most commonly in region close to vertebral column.

The reason for this phenomenon seemingly is based on the anatomical configuration of the base of the lower lobe, and its relationship to the adjacent chest wall and diaphragm. The margin (free edge, lappet) of the lower lobe which separates the thoracic and the diaphragmatic surfaces is for the most part sharp and elongated, and dips deeply into the costophrenic sinus on the anterior, lateral, and posterior aspects of the chest. In the region close to the vertebral column, however, the margin merges with the broad vertical posterior border of the lung and here is blunt and rounded. In this area also, the costophrenic sinus is very shallow. In the presence of an inflammatory reaction beneath the lower lobe, agglutination of the lung margin to the diaphragm in the already mentioned region is often imperfect, because of the smaller and less intimate area of contact between the lung and the structures which constitute the costophrenic sinus. The result is that the enlarging infrapulmonary collection more readily elevates the margin of the lung at this point and comes into contact with the chest wall. It thus, as we say, "acquires a parietal representation."

In the later stages, the enlargement of the empyema takes place as a rule chiefly in the parietal portion, i.e., the part that lies within the general pleural cavity. The end stage of this variety then, discloses the existence of an infrapulmonary collection with a parietal extension of varying size and shape situated usually posteriorly. In some cases the parietal extension remains confined to the lower part of the chest, while in others it rapidly enlarges in all directions and finally may reach such size as to overshadow completely the original infrapulmonary portion (Fig. B).

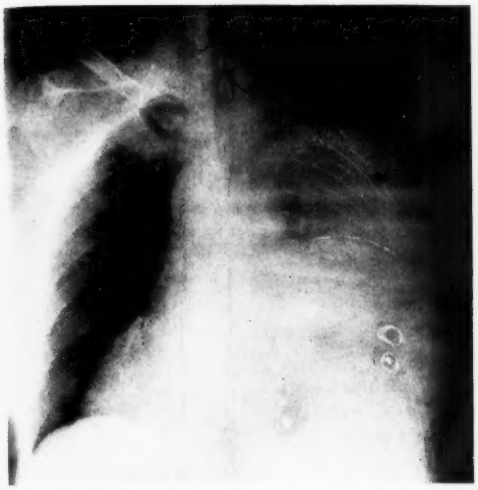

Fig. 3. M.B. Infrapulmonary empyema. Dense shadow with convex upper border occupying major portion of right chest. Base of lung elevated to an extreme degree. Lesion indistinguishable from elevated diaphragm (more advanced lesion than in Fig. 2). Note cardiac displacement. Group 3 lesion.

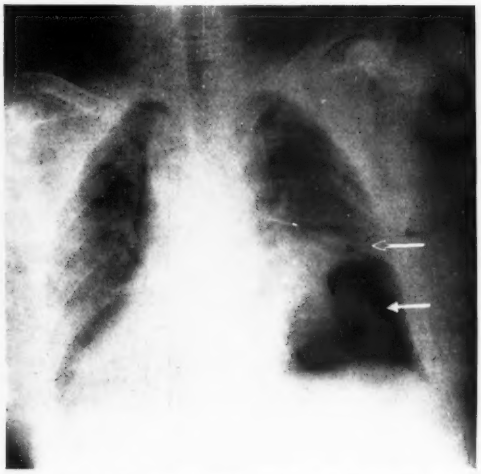

Fig. 4. M.B. Same patient as in Figure 3 several days after operation. Upper arrow indicates base of lower lobe which is still elevated. Lower arrow indicates drained infrapulmonary empyema cavity between base of lower lobe and diaphragm. Note that cardiac displacement is still present.

3. The third group is comprised of cases in which the characteristics of the first and second are combined. The entire effusion remains loculated beneath the lower lobe, but at the same time it is not separated from the chest wall by the margin of the lung. The latter itself becomes elevated due to inadequate primary adhesion formation or to the dissolution of existing adhesions. The result is that the entire under surface of the lobe, including the margin, becomes widely separated from the diaphragm. The collection of pus, therefore, makes contact with the chest wall over a horizontal band-like area between the diaphragm and the margin of the elevated lower lobe. This band-like zone of parietal representation exists usually over the posterior and posterolateral aspects of the chest, since the anterior portion of the lappet as a rule remains adherent in the costophrenic sinus, and therefore does not become elevated (Fig. C).

These in general are the three groups into which the cases fall, but it is to be remembered that the actual extent of the lesion found in each case at operation, will depend on the stage in which the patient is operated upon and the adequacy of the limiting adhesions which tend to keep the process localized.

SYMPTOMATOLOGY

The general symptomatology of infrapulmonary empyema does not differ greatly from that of other varieties of encapsulated empyema. The pleural infection may make itself manifest either during the active course of the pulmonary disease (synpneumonic), or after the pulmonary process has begun to subside (postpneumonic). In the former case, the onset of the pleural infection is apt to be insidious, whereas in the latter case it is usually marked by a recrudescence of such symptoms as cough, fever, malaise, etc.

The symptomatology peculiar to the disease consists in the existence of early pain due to diaphragmatic irritation. As Capps has demonstrated, the site of pain is dependent on the area of diaphragmatic involvement, i.e., central or peripheral, or both. Irritation of the central portion or dome results in pain in the shoulder over the area supplied by the third, fourth, and fifth cervical nerves. This

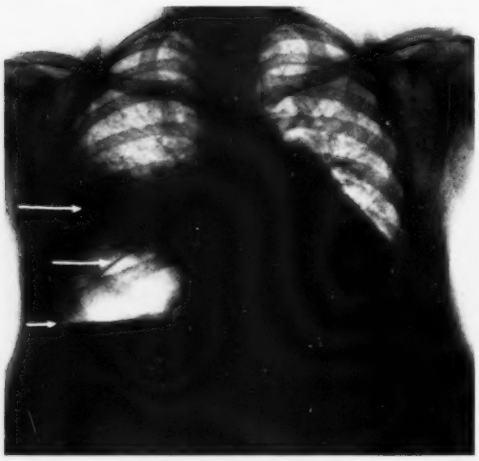

Fig. 5. F.M. Case 1. Infrapulmonary pyopneumothorax. Base of lower lobe (indicated by upper arrow) elevated to a marked degree by a subjacent collection of pus and air. Compressed base of lower lobe at first glance appears to be the diaphragm. (Diaphragm actually lies at a lower level and is obscured by the fluid. [Indicated by the lower arrow]). Note faint lung markings and pulmonary fissure (indicated by center arrow) situated above fluid level, proving that diaphragm lies lower down. Note cardiac displacement. Cardiac enlargement is due to chronic valvular disease. Group 3 lesion.

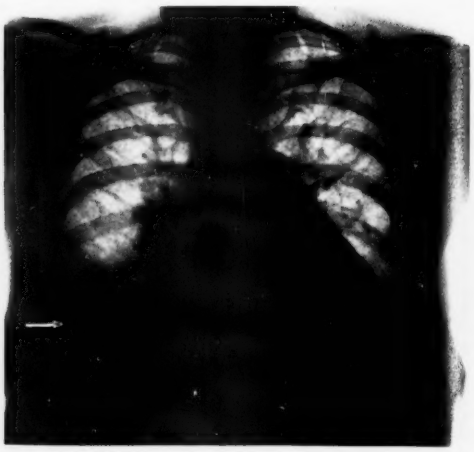

Fig. 6. F.M. Same patient as in Figure 5, 6 months after operation. Note expansion of lung and return of enlarged heart to normal position as indicated by location of its right border. Residual thickening of pleura (indicated by arrow) still present at base.

phenomenon is due to reflex stimulation by way of the phrenic nerve which takes origin from the same roots. Irritation of the peripheral portion of the diaphragm results in pain in the lower chest, upper abdomen, and occasionally even in the lower abdomen. In this connection, Kelly and Weiss have stressed the frequency of pain along the entire costal arch especially posteriorly beneath the twelfth rib. Characteristic pain, if present, is most apt to exist early in the course, before the stage of frank effusion has been reached. As the effusion develops the irritated pleural surfaces become separated by fluid and pain is apt to become less, and often disappears. Since patients are frequently seen late in the course of the infection when pain is absent, the history and site of previous pain may be of great importance.

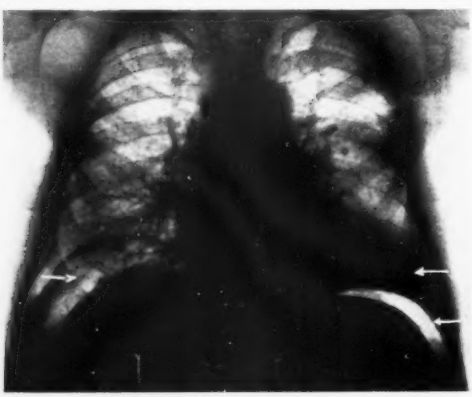

Fig. 7. J.W. Infrapulmonary effusion, left chest. Film taken after induction of pneumoperitoneum to differentiate lesion from a subphrenic abscess. Note air (indicated by arrow on right side and lower arrow on left side) between diaphragm and dome of liver. Effusion (indicated by upper arrow on left side) is present above left diaphragm. Group 1 lesion.

It is to be remembered that pain of diaphragmatic origin may also be noted in the presence of irritative lesions situated beneath, as well as above the diaphragm. It, therefore, is in no sense pathognomonic of infrapulmonary empyema, but rather is to be considered only strongly suggestive, when supported by other findings which will be mentioned later.

PHYSICAL SIGNS

As stated before, an infrapulmonary empyema does not come in contact with the che

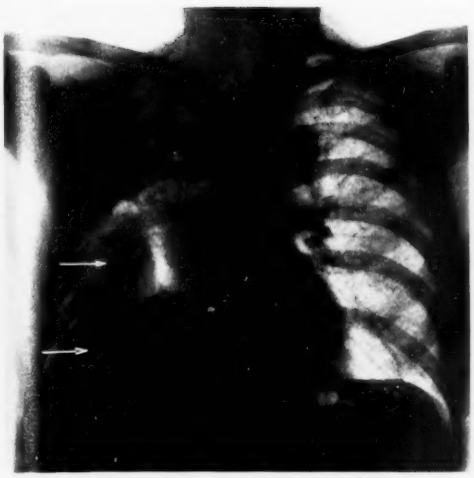

Fig. 8. C.S. Case 4. Infrapulmonary empyema, right chest. Lesion (indistinguishable from elevated diaphragm) is indicated by lower arrow. Moderate sized parietal extension (indicated by upper arrow) extends upward to sixth interspace posteriorly. Note dense infiltration of upper lobe and mesial portion of lower lobe. Note evidence of atelectasis of upper lobe. Group 2 lesion.

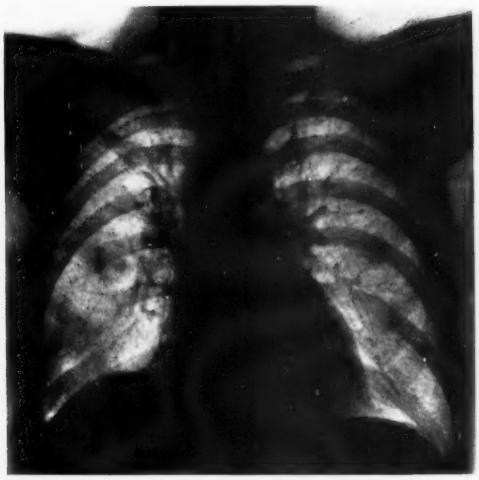

Fig. 9. C.S. Same patient as in Figure 8, 2 months after operation. Note complete expansion of lung and normal appearance of diaphragm. Evidence of chronic hilar tuberculosis present.

wall, when it is completely overlaid on its periphery by the lappet of lower lobe which is adherent to the peripheral portions of the diaphragm (Group 1 under "Pathology and Pathogenesis"). When the lesion is small, the characteristic physical signs of fluid at the base may therefore be absent, and the only signs elicited may be those of the co-existent infiltration in the overlying lung tissue. The percussion note varies from slight dullness to flatness, depending on the size of the purulent effusion. Voice and breath sounds likewise may vary from slight diminution to complete absence. In addition, in the presence of a large effusion, evidence of compression of the base of the lung is present and physical signs of diminished or absent diaphragmatic mobility are usually elicited. Regardless of the nature of the other physical signs, the presence of dullness or flatness at the extreme base should make one suspect the presence of an infrapulmonary empyema, if the patient's clinical course suggests the existence of a suppurative lesion the site of which is not apparent. This point cannot be too strongly emphasized if early diagnosis is to be made. If, in addition, trapezius tenderness is present the diagnosis is more likely.

2. After the collection of pus has extended to the chest wall and acquired a parietal representation (Groups 2 and 3 under "Pathology and Pathogenesis"), the physical signs vary according to the size and shape of the latter. If small, the physical findings do not necessarily become altered, and the only change noted may be the development of localized tenderness to pressure over a small area in the lowermost part of the chest usually close to the vertebral column. This is due to irritation of the underlying pleura as a result of extension of the process to the parietes. As the parietal portion of the collection becomes larger, the signs of an encapsulated empyema in the posterior part of the chest become apparent and often obscure those due to the original infrapulmonary portion.

An infrapulmonary effusion even of moderate size, because of its close proximity to the border of the heart, frequently tends to displace that organ for a variable distance toward the opposite side of the chest. This is particularly true, because of the limited amount of space available for expansion, when the lesion is situated on the left side.

Fig. 10. R.S. Case 7. Infrapulmonary empyema with large parietal extension. Well defined encapsulated empyema of large size (indicated by arrow) is present in lower part of general pleural cavity and extends upward to level of seventh rib posteriorly. Infrapulmonary portion of lesion is completely obscured. Note cardiac displacement. Group 2 lesion.

Fig. 11. R.S. Same patient as in Figure 10, 1 week after operation. Note rapid expansion of lung. Small residual drained cavity (poorly reproduced on film) is indicated by arrow. Residual pleural thickening at base is also still present. Heart has returned to normal position.

The phenomenon, while not constant, occurred with sufficient frequency to attract attention. The four most common causes of cardiac displacement resulting from pleural suppuration are large pleural effusion, tension pyopneumothorax, encapsulation along the mediastinal aspect of the pleura, and parietally situated encapsulation in the lower part of the chest. As a rule these gross lesions can be readily diagnosticated on careful physical examination. In the absence of evidence of their existence, cardiac displacement when associated with dullness or flatness at the extreme base should suggest the diagnosis of infrapulmonary empyema, if the history and clinical course suggest pleural suppuration. In passing it should be stated that elevation of the diaphragm, due to subphrenic or liver abscess, may also at times cause cardiac displacement.

X-RAY EXAMINATION

X-ray examination reveals in general the following groups of findings:

1. When the lesion is small and separated from the chest wall on all aspects by the margin of lower lobe, the shadow cast by the effusion is particularly apt to merge with, and be indistinguishable from, the shadow cast by the overlying infiltrated pulmonary tissue. In cases of this type the most common and in fact the only interpretation that can be made, is that of basilar pneumonia (Fig. 1).

2. Larger, localized infrapulmonary collections usually elevate and arch the base of the lung to a varying degree. As a rule one then sees on the film a dense shadow, with a sharply defined convex upper border, extending across the lower portion of the lung field. This shadow merges below with that of the liver. Because of this fact, the impression may be gained that the picture is one of an elevated diaphragm due to a subphrenic abscess, especially when on fluoroscopy there is noted immobility in the general region of the diaphragm (Figs. 2 and 3). However, the lack of history and physical signs of an intra-abdominal inflammatory lesion which could

be considered responsible for a subphrenic abscess, militates against this interpretation.

3. When the lesion has extended from beneath the lower lobe to the chest wall and then further enlarged in the parietal portion (i.e., the part which lies within the general pleural cavity), the latter rather than the original infrapulmonary portion is often apt to be noted on X-ray examination (Fig. 10). The reason is that the shadow cast by the infrapulmonary portion may become obscured by the shadow of the larger parietal portion. Parietal extensions as stated before, take place generally in a posterior direction, and usually cast readily discernible shadows when films are taken in the antero-posterior erect position. If the parietal portion is small, however, it may be obscured by the superimposed shadow of the infrapulmonary portion or the infiltrated lower lobe. Additional films should, therefore, always be taken with the patient in the lateral erect position, in order to demonstrate these small lesions which lie most commonly in the posterior part of the chest close to the vertebral column.

4. Infrapulmonary pyopneumothorax produces as a rule a characteristic picture (Fig. 5). Extending across the lower part of the lung field one sees a dense, arched, band-like, transverse shadow with a subjacent collection of air surmounting a fluid level. The band-like shadow which at first glance appears to be the diaphragm, represents a portion of the base of the lung which has been elevated and compressed like an accordion, by the collection of air and pus below. The diaphragm is situated at a lower level and is usually obscured by the fluid.

In these cases, the diagnosis of subdiaphragmatic gas-containing abscess may erroneously be made. One feature, however, militates against this interpretation. This is the presence of faint lung markings which are usually seen below the previously mentioned band-like structure that represents not the diaphragm but the elevated portion of the base of the lower lobe. These markings represent the shadows cast by the anterior portion of the lappet of the lower lobe which as previously stated usually becomes adherent in the costophrenic sinus. Another fact that militates against the diagnosis of subphrenic gas-containing abscess, is the lack of history and physical signs suggesting the presence of an intra-abdominal inflammatory lesion or a perforation of the gastro-intestinal tract.

Although not usually necessary, the induction of a small pneumoperitoneum may be of value in cases in which doubt exists as to whether the process is situated above or below the diaphragm. Air when introduced into the peritoneal cavity of a normal person in the erect position, tends to rise and usually collects beneath the diaphragm which it sharply delineates from the dome of the liver below. If the procedure is carried out in a patient with an infrapulmonary empyema or pyopneumothorax, the air likewise collects beneath the diaphragm and delineates the shadow due to the purulent collection and the subjacent diaphragm from the shadow of the liver below (Fig. 7). On the other hand, in a patient with a subphrenic abscess, if a pneumoperitoneum is induced no air enters the space between the diaphragm and the dome of the liver, and the shadows of these structures appear merged. The air then collects below the liver, or in the opposite, uninvolved, subphrenic space, or both. Differential diagnosis is thus made possible.

Cardiac displacement, which is the final point to be considered under X-ray diagnosis, is of the greatest significance when present in those cases in which the films disclose only infiltration in the lower lobe. The infiltration as stated before is usually interpreted as being due to pneumonia. However, since uncomplicated pneumonia is not a cause of cardiac displacement toward the opposite chest and the gross pleural lesions previously enumerated to account for the shifting of the heart are not discernible, the presence of an obscured effusion encapsulated beneath the lower lobe should always be strongly considered (Fig. 1). The importance of this point cannot be overemphasized.

When the infrapulmonary effusion is obvious on X-ray examination, or when parietal extensions are present, cardiac displacement is of interest but of considerably less significance from a diagnostic viewpoint (Figs. 3, 5, and 10).

From the foregoing statements it is evident that correct roentgenographic diagnosis is sometimes difficult or even impossible, especially in the cases in which small or moderate-sized infrapulmonary collections with or without parietal extensions exist. Further procedures are, therefore, sometimes necessary to establish the diagnosis. In this connection the rôle of aspiration of the chest is an important one.

CHEST ASPIRATION

1. Let us consider first the small infrapulmonary collection completely separated from the chest wall by the lappet of lower lobe which is adherent to the peripheral portions of the diaphragm. With this type of lesion, when the aspirating needle is introduced into the chest, pus is not immediately encountered. In order to enter the purulent collection, the needle must be passed through the pulmonary tissue which lies between the chest wall and the empyema cavity. Thus the true nature of the lesion will not be early established by aspiration, even though the physical signs and roentgenographic examination strongly suggest the presence of fluid, unless one is fully aware of the necessity of aspirating the chest deeply.

When a small collection increases in size, the overlying shell of lung interposed between it and the chest wall becomes increasingly thin and more compressed. The aspirating needle then usually encounters pus at a depth only slightly greater than the thickness of the chest wall. Obviously, therefore, the depth to which the needle must be introduced in any given case depends on the thickness of the overlying shell of compressed lung. This in turn depends on the size of the tense encapsulated collection of pus, i.e., the larger the empyema, the thinner will be the overlying shell of lung.

The site of aspiration as a rule is in the ninth or tenth interspace over the posterolateral aspect of the chest, and is best indicated by the height of the basilar shadow on X-ray examination. Attention is called to the possibility of penetrating the diaphragm, by aspiration at too low a level. We have seen this occur in one case, fortunately without untoward effect.

2. When the original infrapulmonary empyema makes contact with the chest wall and a parietal extension develops in the general pleural cavity as demonstrated by X-ray examination, pus will be encountered as soon as the needle enters the chest, provided the aspiration is performed at the proper site. This point is best indicated by a study of the films taken in both the anteroposterior and lateral erect positions.

3. When the history and physical findings suggest the presence of infrapulmonary suppuration although X-ray examination reveals only infiltration in the lower lobe, aspiration should be deferred until such time as the presence and extent of the effusion become apparent on X-ray examination. On the other hand, immediate exploratory puncture may very occasionally be indicated because of the patient's progressively downhill course as the result of marked toxic absorption.

It should be emphasized that once pus has been encountered in the case of a collection loculated beneath the lower lobe, the needle should not be withdrawn, but should be left *in situ* and operation proceeded with as soon as possible. This is of prime importance, because the collection may be so small or difficult of access that, after the needle has been withdrawn, later attempts to locate it may fail. It follows, therefore, that aspiration should be performed only when operation can be proceeded with promptly.

DIAGNOSIS

The diagnosis of infrapulmonary empyema rests on a complete consideration of the history, physical signs, clinical course, X-ray examination, and the result of aspiration of the chest.

As has been stated before, the history is not characteristic and can at best only suggest the presence of suppuration within the chest. If pain of diaphragmatic origin exists, or formerly existed, the diagnosis is more likely. Depending on the size of the effusion, the physical signs of fluid at the base may be definite in some cases and equivocal in others. When the lesion is of fair size, X-ray examination usually reveals its presence, whether or not frank physical signs of fluid exist at the base. The

effusion may be clearly demonstrated to be situated beneath the lower lobe. At times, however, the induction of artificial pneumoperitoneum is necessary to distinguish it from a subphrenic or liver abscess. The latter is also usually excluded by the history and clinical course.

When frank physical signs of fluid at the base are not present, one usually elicits signs of consolidation in the lower lobe. In these cases, X-ray examination usually reveals only infiltration which is interpreted as being due to pneumonia in the lower lobe. In other cases, even when the physical signs of fluid are definite, the X-ray examination may reveal identical findings.

Regardless of whether the physical signs are interpreted as being due to consolidation or fluid at the base, a history and clinical course suggesting the existence of suppuration within the chest, the site of which is not apparent, should always arouse suspicion. Even an X-ray examination which reveals only the presence of infiltration in the lower lobe, does not militate against the diagnosis. The coexistence or history of tenderness along the trapezius or over the lower chest posteriorly, lends support to the diagnosis. If, in addition, cardiac displacement toward the opposite side of the chest without apparent cause is present, the diagnosis is practically certain.

In late cases, X-ray and physical examination may disclose a parietally-situated empyema in the lower part of the general pleural cavity, which only at operation can be proved to be either a simple encapsulated collection or a parietal extension of an original infrapulmonary lesion.

Aspiration is merely confirmatory in those cases in which the effusion is demonstrated on X-ray examination. When the X-ray films are negative for fluid, however, it is truly an exploratory procedure which is sometimes necessary to confirm or disprove the presence of a suspected collection of pus beneath the lower lobe. The necessity for aspirating deeply in certain cases is again emphasized.

TREATMENT

1. In those instances in which the collection of pus has extended from beneath the lower lobe to the chest wall and therefore has acquired a parietal representation (Groups 2 and 3 under "Pathology and Pathogenesis"), the plan of procedure is to excise a segment of overlying rib and to enter the cavity safely within the area of limiting adhesions. The rib is then further removed anteriorly and posteriorly, in order adequately to expose the pocket to its limits. If necessary, the intercostal musculature above and below the resected rib may be excised and segments of adjacent ribs removed, in order not only to afford an adequate view of the existing pathological process but also to facilitate drainage. Care must always be exercised to remain within the area of limiting adhesions and thus to avoid infecting the free pleural cavity. After all pus and detritus have been evacuated by suction, a sterile examining light is introduced and the interior of the cavity inspected.

Free exposure and direct inspection are of prime importance in all suspected cases of infrapulmonary empyema. As stated before, if a patient is first seen in the later stages of illness with a large parietal empyema, it is often impossible to determine whether one is dealing with the common variety of parietal empyema or a large parietal extension from an original infrapulmonary collection. If intercostal drainage or short rib resection without inspection of the interior of the cavity is performed, one is still unable to answer the question. Of more serious import is the danger of inadequate drainage of the primary pleural focus, which may result if an infrapulmonary pocket is present. Drainage is particularly apt to be poor in the cases in which the parietal and infrapulmonary pockets communicate by means of a narrow tract (see Case 8 of abstracts). Furthermore, in cases of putrid empyema due to infection by anaerobes complete unlocking of the suppurative focus with resultant free aeration is the only method which can be depended upon to lead to prompt subsidence of infection.

The next step in the procedure is to inspect the interior of the infrapulmonary pocket and to seek the site of entry of the pleural infection. This is of great importance, as failure to recognize and properly treat the underlying pulmonary lesion, especially in cases of rup-

tured putrid lung abscess, may result in chronicity of the lesion itself, with or without later recurrence of the empyema even after the wound has healed. If the pleural infection has been the result of seepage or the perforation of a small subpleural abscess, no additional procedure is required. On the other hand, if there has been a small or inadequate perforation of a comparatively large pulmonary focus, it is imperative that the lesion be adequately laid open to insure satisfactory drainage. If this is not done, the small perforation may become temporarily sealed over, only to result in activation of the lesion at a later time. Early in our experience this course of events was noted on several occasions in cases of perforated putrid lung abscess, when the resultant putrid empyema involving the general pleural cavity had been evacuated without visualizing and treating the underlying pulmonary lesion. Several of the patients at a later date had to undergo further operative procedures for the treatment of the lung abscess which was originally present.

After the pulmonary focus has been adequately opened, the entire empyema cavity and the pulmonary focus are packed with iodoform gauze to the limits of all recesses.

2. In those instances in which the empyema is situated entirely beneath the lower lobe and separated from the chest wall on all aspects by the lappet of the lobe which is adherent to the periphery of the diaphragm (Group 1 under "Pathology and Pathogenesis"), the free pleura may or may not have to be traversed in order to drain the collection.

It has been stated before that usually in the presence of medium or large sized collections, the overlying pulmonary tissue that separates the empyema from the chest wall, is quite thin. Furthermore, as a result of the juxtaposition of the infrapulmonary suppurative process, this shell of lung becomes involved by an inflammatory reaction which usually results in its firm agglutination to the adjacent chest wall, as well as to the diaphragm. Under such circumstances, if the operative incision is properly placed, free pleura is not entered, and the collection of pus is at once encountered after the chest wall and the underlying thin shell of adherent lung are traversed.

On the other hand, if the collection is small, there may be no widespread overlying peripheral inflammatory reaction and the peripheral portions of lower lobe may be adherent only to the diaphragm but not to the chest wall. This means that the free pleura will have to be traversed, and the adherent lappet detached from the diaphragm, in order to establish drainage. In these cases the operative approach is made low in the chest at the site of the inlying aspirating needle, a segment of rib then excised, and the free pleura entered. The adjacent free pleural cavity is next packed off with gauze. If the pus which had previously been withdrawn into the aspirating syringe is odorless, the assumption that the lesion is not a putrid anaerobic infection is justifiable. One may then proceed to peel the adherent lappet of lower lobe off the diaphragm and evacuate the empyema. The pulmonary lesion is inspected, treated as described if necessary, and the cavity packed. Care must be exercised not to displace the gauze which is walling off the free pleural cavity.

In those instances in which foul pus had been previously aspirated, the underlying source of the infection is either a ruptured putrid lung abscess or a ruptured, anaerobically infected bronchiectatic cavity. Because of the danger of widespread, virulent, anaerobic pleural infection in these cases, and the fact that the underlying pulmonary lesion must be adequately exposed in order to be properly treated, it is advisable if no parietal adhesions exist, to perform the operation in two stages. At the first stage, after the free pleura has been entered, the margin of lower lobe and the adjacent lung are shut off from the free pleura by packing, or by suturing the open edges of the wound down to the lung about the operative field. The wound is then widely packed with gauze to stimulate the formation of adhesions, and thus to assure complete shutting-off of the free pleura. When this has been accomplished the second stage is performed, the empyema evacuated, and the pulmonary lesion treated as described. In the extremely rare instances in which the patient's precarious condition, due to marked toxic absorption, renders the evacuation of pus urgent, the two stages may be combined and performed at one operation.

From the foregoing statements in regard to the drainage of collections which are situated entirely beneath the lower lobe, it is evident that the larger the lesion, the simpler is the operative problem. It is, therefore, always wise, if the patient's condition permits, to delay operation until such time as the lesion has reached large size or a parietal extension has developed. Both of these eventualities permit the operation to be performed in one stage. In the event, however, that the patient's condition makes operation imperative, one should proceed immediately. This is neces-

sary most often in cases in which anaerobic organisms, that give rise to virulent putrid infections, are present.

3. In some instances as previously stated, certain features of the history, clinical course, and physical examination warrant the assumption that an infrapulmonary collection exists, even though the X-ray examination is not conclusive and exploratory aspiration does not disclose it. If the patient is observed for a sufficient period of time, the lesion usually becomes more evident due to an increase in size. Expectant treatment is, therefore, advisable until this occurs. On the other hand, in the rare instances in which the lesion does not become more obvious and the grave condition of the patient does not warrant further observation, exploratory operation may very occasionally be indicated. The operative approach is made over the posterior aspect of the lower chest at a level best indicated by the height of the diaphragm. A segment of rib is removed and aspiration performed. If repeated aspiration is negative, the pleura is opened and the further plan of procedure, as described, is followed.

The postoperative treatment in practically all cases consists in changing the packings periodically until the cavity becomes obliterated by the expansion of the lung. The first dressing is usually done at the end of about a week, unless there is evidence of retention within the wound or the discharge is unusually profuse. Repacking of the wound is performed under direct vision, retractors being used to obtain full exposure, and a sterile examining light to afford adequate illumination. Care is taken to pack the cavity to its limits and thus to avoid the shutting off of infected recesses and the irregular expansion of the lung. Subsequent dressings are done at intervals of about 4 or 5 days and the lung is allowed to expand gradually and evenly. When the cavity has shrunken to small size, all packings are left out, and the wound is allowed to heal. The general care differs in no respect from that of patients with empyema of the usual variety.

Obliteration of the empyema cavity is usually prompt, and secondary procedures in the event of slow healing have not been found necessary. In the event of slow obliteration, however, crushing of the phrenic nerve through a cervical approach suggests itself, provided the diaphragm is mobile. This procedure causes the diaphragm to rise as a result of temporary paralysis, and thus tends to decrease the size of the dead space. Within a period of several weeks or months, during which time the cavity becomes obliterated, the mobility of the diaphragm slowly returns and eventually again becomes normal.

RESULTS OF TREATMENT

Of the 12 patients, 11 were treated surgically in the general manner described. In the other instance (Case 9) after the removal of a foreign body from the right lower lobe bronchus, the pulmonary process subsided, and following the aspiration of an infrapulmonary collection of thin pus, the effusion did not re-accumulate.

In the 12 cases, there were no deaths attributable to the pleural infection or to the procedure employed. One patient (Case 10) with a chronic putrid lung abscess which ruptured, died at a later date with the picture of metastatic cerebral involvement from the original lung abscess. At the time of death, the empyema cavity was healing and was considered to be in no way related to the fatal termination. The 11 remaining patients made uncomplicated recoveries.

ABSTRACTS OF CASES

CASE 1. F.M., female, aged 39 years, November 2, 1931 to December 13, 1931. Diagnosis: Chronic cardiovalvular disease. Auricular fibrillation. Pulmonary infarction with necrosis, resultant infrapulmonary pyopneumothorax.

Patient's history was typical of pulmonary infarction with development of pneumonitis in the right lower lobe. X-ray examination on admission revealed the presence of infiltration in the lower lobe and infrapulmonary pyopneumothorax (Fig. 5). Because of the patient's precarious condition as a result of the pleuropulmonary infection together with cardiac decompensation, preliminary closed drainage was performed, according to the author's method (7, 8). Twenty-three days later, thoracotomy, rib resection, and drainage were performed, after the general condition had improved. A large necrotic infarct involving the base of the lower lobe together with a pyopneumothorax were found. The lower lobe was widely elevated. Convalescence was uneventful (Fig. 6. Group 3 lesion).

CASE 2. A.P., female, aged 14 years, December 14, 1929 to January 30, 1930. Diagnosis: Postpneumonic infrapulmonary empyema.

History was typical of pneumonia of right lower lobe. Clinical course after crisis suggested empyema. X-ray examination revealed only infiltration in the lower lobe. Repeated aspiration was negative; at the end of 24 days finally positive. At operation, infrapulmonary empyema with small parietal extension was found. Bacteriology: pneumococcus type 1. Convalescence was uneventful. Group 2 lesion.

CASE 3. C.F., female, aged 54 years, January 3, 1930 to January 28, 1930. Diagnosis: Postpneumonic infrapulmonary empyema.

Patient's history was typical of pneumonia of the right lower lobe. The clinical course after crisis suggested empyema. X-ray examination revealed only infiltration in the lower lobe. Aspiration of chest was positive. At operation a ruptured subpleural pneumococcus type 2 abscess was found on the under surface of the lower lobe. Infrapulmonary empyema with small parietal extension was present. Convalescence was uneventful. Group 2 lesion.

CASE 4. C.S., male, aged 44 years, March 28, 1930 to May 6, 1930. Diagnosis: Postpneumonic infrapulmonary empyema. Patient's history was typical of extensive lobar pneumonia of the right upper and lower lobes, associated with atelectasis. Subsequent course was suggestive of empyema with signs of fluid posteriorly. There was clinical evidence of atelectasis, i.e., displacement of trachea and heart toward the involved side. Diaphragm appeared elevated (Fig. 8). At operation, infrapulmonary empyema with parietal extension (posteriorly) was found. Appearance of elevated diaphragm was due to infrapulmonary collection. Bacteriology: pneumococcus type 3. Convalescence was uneventful (Fig. 9). Group 2 lesion.

CASE 5. J.D., female, aged 34 years, January 18, 1932 to February 28, 1932. Diagnosis: Acute putrid lung abscess with rupture, and secondary infrapulmonary empyema. Patient said she had suffered a sudden onset of pain in the lower chest, with dyspnœa. There was an early development of signs of fluid, and the course was septic. X-ray examination revealed only consolidation of lower lobe. Repeated aspiration was negative but finally positive after 10 days. At operation an infrapulmonary empyema with small parietal extension was found. The underlying pulmonary lesion apparently was a small putrid abscess, without known etiology (6). Bacteriology: gram positive anaerobic bacillus. Convalescence was uneventful. Group 2 lesion.

CASE 6. L.W., male, aged 48 years, September 15, 1928 to November 6, 1928. Diagnosis: Chronic putrid lung abscess with rupture, and putrid infrapulmonary empyema.

There was X-ray evidence of a small empyema at the extreme base posteriorly, which was noted only on lateral films. At operation, a small infrapulmonary empyema with parietal extension was found and evacuated. Bacteriology: mixed anaerobes. Convalescence was uneventful. Subsequent drainage of multiple lung abscesses at other sites was carried out. Group 2 lesion.

CASE 7. R.S., female, aged 40 years, February 1, 1930 to March 18, 1930. Diagnosis: Postpneumonic infrapulmonary empyema.

X-ray examination disclosed only a large empyema in the posterior part of the chest (Fig. 10). At operation, this was proved to be a large parietal extension of an original infrapulmonary empyema. Bacteriology: pneumococcus type 2. Convalescence was uneventful. (Figure 11 was taken after operation). Group 2 lesion.

CASE 8. W.B., male, aged 20 years, April 19, 1930 to May 11, 1931. Diagnosis: Chronic postpneumonic infrapulmonary empyema. One year before, patient was operated upon at another hospital for left-sided empyema. The wound healed in 3 months, but subsequently re-opened and drained on several occasions.

At operation, an infrapulmonary empyema was found communicating by means of a narrow tract with an area of parietal extension. The lesion was widely exposed and packed. Bacteriology: pneumococcus type 4. Convalescence was uneventful.

The case illustrates the evolution of a chronic empyema due to the presence of an unsuspected, inadequately drained infrapulmonary pocket which was not discovered at the time of original operation because of inadequate exposure. This lesion caused infection to be maintained in the parietal extension, with failure of healing. Group 2 lesion.

CASE 9. L.I., male, aged 38 years, November 19, 1931 to January 13, 1932. Diagnosis: Synpneumonic infrapulmonary empyema. History of aspiration of foreign body (chicken bone) 11 weeks previously. Subsequent development of pneumonia of right lower lobe due to occlusion of lower lobe bronchus by the foreign body. Then the development of infrapulmonary empyema. X-ray with induction of pneumoperitoneum was necessary to differentiate the lesion from a high diaphragm.

The foreign body was removed bronchoscopically. On aspiration of chest, effusion was found to be thin and cloudy. It contained 14,000 leucocytes per cubic millimeter with 80 per cent polymorphonuclears. Smear and culture were negative. Following removal of the foreign body, the pneumonitis cleared and effusion did not recur. Group 3 lesion.

CASE 10. H.K., male, aged 42 years, February 2, 1932 to March 6, 1932. Diagnosis: Chronic putrid lung abscess with rupture, and putrid infrapulmonary empyema.

The lesion was demonstrated by the X-ray. A posterior empyema was also seen. After the posterior empyema was drained, the infrapulmonary

collection was later evacuated by peeling the lappet of lower lobe, from the diaphragm. The lung abscess which was the cause of the lesions was found in the lappet. It had ruptured in two directions giving rise to the two empyemas, which were anatomically separated from one another. Bacteriology: mixed anaerobes. Patient died at a later date with the picture of metastatic cerebral involvement. Group 1 lesion.

CASE 11. C.D., male, aged 54 years, April 16, 1932 to May 25, 1932. Diagnosis: Postpneumonic infrapulmonary empyema. The history was typical of lobar pneumonia followed by clinical course of empyema but with signs interpreted as being due only to infiltration in the lower lobe (Fig. 1). Later these developed signs of fluid posteriorly at base adjacent to vertebral column, and the presence of fluid was verified by the X-ray. Operation revealed a small infrapulmonary empyema, with a large parietal extension. Bacteriology: pneumococcus type 3. Convalescence was uneventful. Group 2 lesion.

CASE 12. D.H., female, aged 15 years, June 11, 1932 to July 14, 1932. Diagnosis: Postpneumonic infrapulmonary empyema. The diagnosis was made on the basis of the clinical course which suggested intrapleural suppuration. There were signs of dullness at the base, despite several negative chest aspirations. X-ray examination revealed cardiac displacement and a shadow in the lower chest, the exact site of which was not entirely clear. After drainage of the infrapulmonary empyema, a parietal empyema which was entirely separate from the former was later found and evacuated. The causative pulmonary lesion, a ruptured subpleural abscess was situated in the lappet of lower lobe and had ruptured in two directions giving rise to the two separate empyemas. (Same as in Case 10, H.K.) Bacteriology: pneumococcus type 1. Convalescence was uneventful. Group 1 lesion.

SUMMARY

1. Infrapulmonary empyema is a variety of encapsulated empyema interposed between the under surface of the lower lobe of the lung and the diaphragm.

2. The essential feature of the pathogenesis in the group of cases under discussion, is the presence of an infective lesion involving the basilar portion of the lower lobe with the subsequent development of a suppurative process in the subjacent infrapulmonary pleural space.

3. The organisms present vary according to the type existing in the underlying pulmonary source of infection.

4. The cases are divided into three groups and each group described.

5. The symptomatology, physical signs, X-ray findings, diagnostic methods, and differential diagnosis are discussed.

6. The significant features in the diagnosis of early cases are:

a. History and clinical course suggesting intrapleural suppuration, the site of which is not apparent.

b. Co-existence or history of pain and tenderness of diaphragmatic origin.

c. Physical signs of infiltration in the lower lobe, with or without signs suggesting fluid.

d. X-ray evidence of infiltration in the lower lobe without evidence of pleural effusion, and sometimes with cardiac displacement.

e. Negative result on superficial chest aspiration.

7. The surgical treatment of the various types of infrapulmonary empyema is described.

8. The results of treatment are presented.

9. Twelve abstracted case histories are presented.

The author wishes to express his indebtedness to Dr. Harold Neuhof for his helpful suggestions and constructive criticism.

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A STUDY OF THE VESICAL END OF THE URETER IN HYDRONEPHROSIS[1]

A Report of Fifteen Cases

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WHEN infants and children are examined and pyelograms, after either intravenous injection or the injection of opaque media from below, are made and show dilatation of the kidney pelvis and ureter, a diagnosis of stricture of the ureter with hydronephrosis and hydroureter is suggested.

Because of the general interest in the subject of stricture of the ureter in adults, and because hydroureter with hydronephrosis is frequently found in children, we became interested in determining whether or not in these cases the obstruction at the lower end of the ureter was due to stricture in the generally accepted interpretation of that word.

According to Braasch and Frater, "a stricture is a narrowing, beyond the normal anatomical and physiological limits, of a hollow muscular tube. Microscopically a stricture may show: (a) changes in the epithelium, such as destructions; (b) narrowing of the lumen to complete obliteration; (c) evidence of inflammatory reaction, such as leucocytic collections in the wall of the ureter; (d) increase in fibrous tissue normally present; and (e) hyalinization of muscle."

The histological study in our cases, instead of showing the foregoing requisites for stricture, showed that the obstruction which produced the hydroureter and hydronephrosis was due to hypertrophy of the muscular longitudinal fibers; with few exceptions to anomalous insertions of the ureters. Inflammatory changes were of recent origin only, and were not the cause of the essential primary narrowing of the lower ends of the ureters.

This paper is based on a series of 15 postmortem specimens from the Children's Memorial Hospital of Chicago.

Case 1.[2] R. C., male, aged 1 month, admitted to the Children's Memorial Hospital, September 12, 1927, on account of projectile vomiting. There were no urinary symptoms. The physical examination was negative except for a slight injection of the throat and a questionable walnut-sized mass in the right upper quadrant. Peristaltic waves were visible. The urinalysis was negative. A tentative diagnosis was made of pyloric obstruction and operation was advised. On September 12, 1927, an exploratory laparotomy was performed. No pyloric tumor was found. Temperature varied from 100 to 102 degrees following the operation. The child died on the second day following the operation. The clinical diagnosis was bronchopneumonia. An autopsy was done on September 15, 1927. The anatomical diagnosis was congenital valve in the posterior urethra; hypertrophy and dilatation of the urinary bladder; hydroureter and hydronephrosis, bilateral, with infection (streptococcus); and a bronchopneumonia.

Description of specimen. The kidneys show a moderate amount of hydronephrosis. The renal pelves are dilated. The ureters are dilated from the renal pelves down to the bladder wall. The degree of dilatation is as marked at the bladder as at the upper end. The wall of the bladder is enormously dilated, but there are no evidences of trabeculation. The ureteral orifices appear normal.

The intramural part of the ureter appears thick and rigid. Further examination shows that the dilatation begins at the juxtavesical part of the ureter. The ureters are enormously dilated and tortuous, and the walls are thin. When the dissection of the ureters is carried through the wall of the bladder, it is noted that the intramural parts of the ureters are thickened but not dilated.

Histology. In serial sections of the juxtavesical portion of each ureter, the lumen is contracted and has thick epithelium of long cells, and a rather narrow mucous membrane. At least three-fourths of the ureter wall consists of muscle fibers, most of which are longitudinal, and only a few are circular fibers. The muscle is in small bundles separated by delicate fibrous reticula.

The serial sections of both ureters are essentially identical. Muscle hypertrophy is marked and in the lower ureter distal to the juxtavesical portion the circular and longitudinal fibers are about equal in amount.

In the midportion of the ureters their walls are thin, the lining epithelium is narrow, the mucous membrane practically obliterated, and the structure is largely muscular, circular and longitudinal fibers being equally distributed. The peripheral connective tissue is scant.

[2] Previously reported in "Congenital valves of the posterior urethra." (Kretschmer and Pierson). Am. J. Dis. Child., 1928, xxxviii, 804.

[1] From the Children's Memorial Hospital and from the A. D. Thompson Urological Fund of Rush Medical College of the University of Chicago. Read at the Annual Meeting of the American Association of Genito-Urinary Surgeons, May 26, 27, and 28, 1932, at Niagara Falls, Canada.

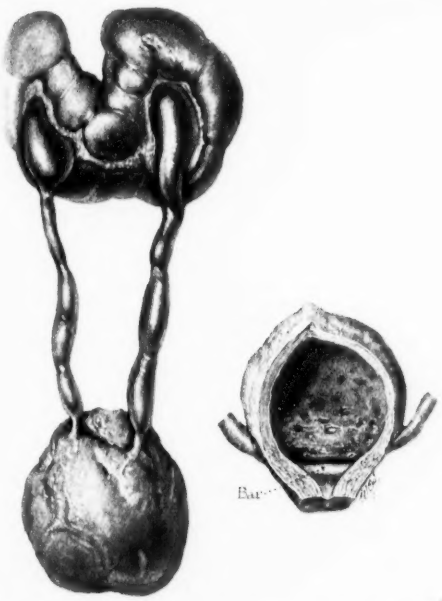

Fig. 1. Case 2. Horseshoe kidney with bilateral dilatation of kidney pelves and ureters. The dilatation terminates about 5 centimeters above the bladder. Bladder wall thickened. Note presence of bar.

There is extensive hyalinization of the bladder musculature. The bladder wall is only slightly thicker than the wall of the ureter.

CASE 2. C. M., male, aged 1 year, admitted March 18, 1931. The infant had a spina bifida and a painless swelling of the left thigh and left leg of 1 week's duration. When born there was a swelling on the back about the size of half an egg, and this discharged for 2 or 3 months. There was marked incontinence of urine. Examination revealed a pale, fat, and apparently ill infant. There was marked swelling of the left lower extremity from the hip to and including the foot; marked bilateral calcaneo-equinovalgus. Knee jerks were absent. Roentgen-ray examination revealed fracture of the left femur. The child ran a septic temperature which rose to 105 degrees daily. Examination of the urine showed albumin and a small amount of pus. Subsequently bilateral otitis media and bronchopneumonia developed and the child died on April 15, 1931. The clinical diagnosis was spina bifida; talipes equinovalgus; fracture of the femur; bronchopneumonia; otitis media; secondary anæmia. Autopsy was done on April 16, 1931. The anatomical diagnosis was horseshoe-shape kidney, hydroureters and hydronephrosis; extensive bilateral bronchopneumonia; spina bifida; talipes equinovalgus; fracture of the femur; generalized anæmia; acute otitis media.

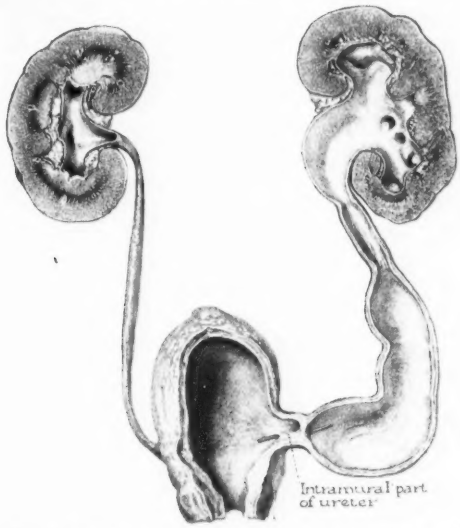

Fig. 2. Case 3. The left ureteral orifice is normal; intramural part of the ureter not dilated. Dilatation of ureter begins above the bladder. Note presence of superficial diverticulum.

Description of specimen. The kidneys are completely fused at the lower poles and have a separate blood supply and each half has a separate pelvis (Fig. 1). Both renal pelves are equally dilated.

The ureters are dilated, the left more so than the right. The dilatation extends from the ureteropelvic junction down to a point about 5 millimeters above the bladder on the right side and about 4 millimeters above the bladder on the left side, at which point the ureter is of normal circumference and thickened. Below this point the ureter approaches the normal and the intramural part is normal.

The bladder wall is thickened and shows some trabeculations with superficial cellules and the presence of a median bar. The ureteral openings are normal.

Histology. In serial sections of the lower end of each ureter, the walls are thick, the lumen contracted and lined with stratified epithelium in folds. There is practically no subepithelial mucous membrane. The thickness of this contracted portion is composed essentially of muscle fibers, the longitudinal fibers predominating. These fibers are encased in connective tissue extending to the periphery.

Directly above the juxtavesical portions the lumen is larger, its diameter being at least twice as great as below. Here the muscle hypertrophy is marked, but the fibers are farther apart and the connective tissue less dense.

Similar serial sections of a portion of the midureter and upper ureter are thin. This thinness is in all the layers. The mucous membrane is almost absent.

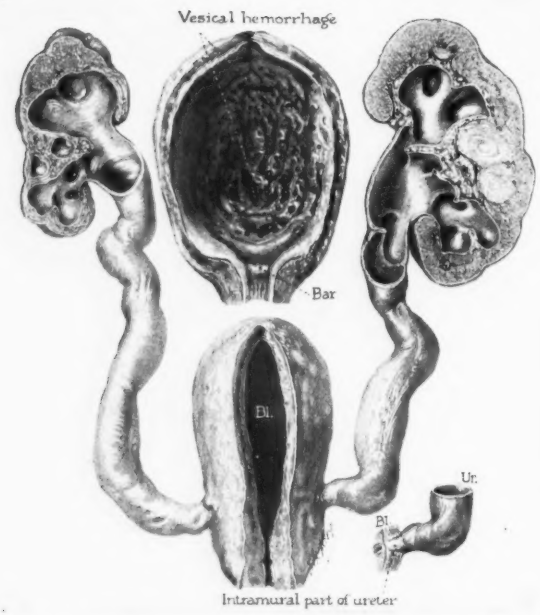

Fig. 3. Case 4. Enormous dilatation of the kidney pelves and ureters down to the bladder wall. Intramural part of the ureter not dilated. Insert, Thickening of trigone and bladder wall. Presence of median bar.

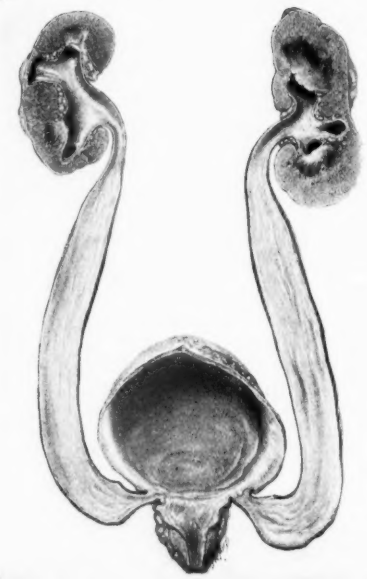

Fig. 4. Case 6. The ureters were uniformly thickened and dilated nearly to the bladder wall.

CASE 3. T. S., male, aged 12 months, admitted September 2, 1925. The following symptoms: intense restlessness, fever, diarrhœa, vomiting, and loss of weight, began 1 week prior to admission to the hospital. Urinary symptoms were absent. Examination revealed a well developed, dehydrated infant. Physical examination was negative. Temperature on admission was 102.6 degrees. Examination of the urine showed a large amount of pus and albumin, and Bacillus coli on culture. Death occurred 16 hours after admission to the hospital. The clinical diagnosis was alimentary intoxication. Autopsy was done September 3, 1925. The anatomical diagnosis was emaciation and generalized anæmia; hydroureter and hydronephrosis, left; ectopic right kidney.

Description of specimen. Fetal lobulations are noted in the right kidney; otherwise normal (Fig. 2). The right ureter measures 85 millimeters in length. A slight evidence of dilatation is noted in the left kidney. The left ureter from its orifice to the ureteropelvic junction measures 175 millimeters. It is not tortuous, the walls are thickened and dilated. The point of maximum dilatation, 35 millimeters in circumference, is about 30 millimeters above the ureteral orifice. Below this point of maximum dilatation the ureter is 4 millimeters in diameter; its intramural part is apparently normal. The bladder wall shows well defined hypertrophy and the presence of a small diverticulum at the site of insertion of the left ureter into the bladder. The autopsy records of this case fail to state whether or not obstruction was present at or in front of the neck of the bladder.

Histology. The serial sections of the lower portion of the left ureter contain a part of a bladder diverticulum behind which is the narrow ureter. The epithelium here is narrow, and generally only two cells high. The mucous membrane varies in amount but is generally thick and extends centrally into the folds of the epithelium, being well defined peripherally where it joins the muscularis. Here the muscle bundles, each bundle consisting of compressed fibers, are clumped together. These bundles are generally three to five times the usual size, and about two-thirds of them consist of longitudinal fibers. In cross sections, portions of the wall are without muscle fibers between the lumen and periphery, and it is in the periphery that the fibers are clumped. In serial sections higher up in the ureter the wall is thin, the epithelium narrow, the mucous membrane absent, and the essential content of the wall is the muscularis. Throughout all sections of the ureter there is a rather extensive infiltration of lymphocytes into the lining mucous membrane.

CASE 4. D. W., male, aged 4 years, admitted March 8, 1919, on account of fever, cough, loss of appetite, and constipation. Incontinence of uri

Fig. 5.

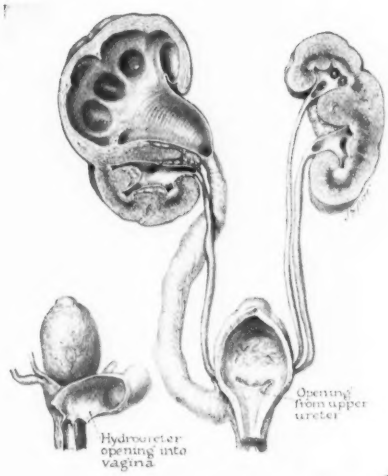

Fig. 6.

Fig. 5, left. Case 7. Note thickening of intramural and juxtavesical part of the ureter with dilatation above for about 45 millimeters. Thickening of wall of ureter above this part.

Fig. 6. Case 8. Note enormous dilatation of the ureter draining the upper half of the right double kidney. Insert, Showing insertion of ureter into the vagina.

was noted on admission; the child was wet all the time. Temperature, 102.4 degrees. The child appeared to be acutely ill. There was a slight discharge from the nose. The tonsils were large and covered with a yellow mucous material. The lungs revealed many mucous râles, posteriorly from the third to the ninth ribs on both sides. In the abdomen was a suprapubic tumor which disappeared after catheterization, but on the following day the tumor reappeared. After each catheterization the tumor disappeared. Examination of the blood showed white cells 17,000; red cells 5,150,000; hæmoglobin 70 per cent. Examination of the urine showed pus and Bacillus coli on culture. Death occurred on March 31, 1919. The clinical diagnosis was acute pyelocystitis; obstruction at the neck of the bladder; retention of urine. Autopsy was done March 31, 1919. The anatomical diagnosis was multiple abscesses of the kidneys; bilateral hydronephrosis with infection; dilatation of ureters; median bar; chronic cystitis; hypertrophy of bladder wall; congenital valve in posterior urethra.

Description of specimen (Fig. 3). The left kidney is three times the size of the right; the surface is studded with many miliary abscesses. A moderate amount of hydronephrosis is noted. The right kidney is almost completely destroyed by hydronephrosis. Only a small amount of renal tissue remains at the upper pole. The ureters are dilated, very tortuous, and the walls are decidedly thickened. The dilatation extends from the ureteropelvic junction down to the bladder. The intramural part of the ureter is thickened but not dilated. No evidence of narrowing of the lumen is noted. The dilatation begins above the intramural part of the ureter. The bladder wall is thick, the mucosa rough and markedly hyperæmic, in places mottled with recent hæmorrhages; most marked on the back portion. The interureteric ligament is thickened. The ureteral openings are normal. A median bar and a congenital valve in the posterior urethra are present.

Histology. Serial sections were studied from fourteen blocks of different portions of the ureters. The lining mucous membrane was swollen in all of them and was the site of subacute and chronic inflammation with a purulent exudate into the lumen. From the intramural portions distally to the kidneys the changes of both ureters were identical. In the intramural, and especially in the juxtavesical portions of the ureters, the muscle fibers were so greatly enlarged that in places the thickened wall had muscle fibers for four-fifths of its thickness. The longitudinal fibers were slightly more abundant than the circular ones, many of which had undergone hyaline degeneration. Everywhere the mucous membrane was narrow.

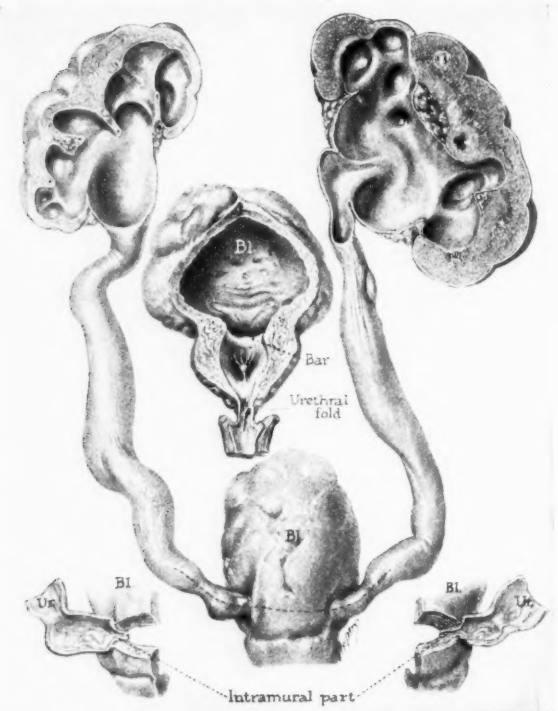

Fig. 7. Case 9. Inserts showing dilatation of ureters down to the bladder wall. Intramural part of ureters thickened but not dilated. Note median bar and valve in urethra. Trigone thickened.

As the ureters were followed to the kidney the wall became less thick, the predominance of circular and longitudinal fibers varied and were more nearly normal in amount.

Case 5. D. S., female, aged 32 days, was first admitted November 5, 1928. The significant findings at that time were otitis media and a prolapsed rectum together with exstrophy of the bladder. Examination of the urine showed a few pus cells only. On second admission, March 19, 1929, the child was admitted because of a prolapsed rectum and an acute attack of pyelitis. The infant subsequently developed bronchopneumonia and died on March 26, 1929. The clinical diagnosis was exstrophy of the bladder; acute pyelonephritis; prolapse of the rectum; otitis media; bronchopneumonia; empyema, right. Autopsy was done March 26, 1929. The anatomical diagnosis was exstrophy of the bladder; subacute cystitis and ureteritis; diffuse bronchopneumonia; empyema; ventral hernia.

Description of specimen. The urinary bladder is present in the front abdominal wall in the midline just above the symphysis pubis for a place 4.5 centimeters in diameter. It is sharply defined from the adjacent, pale white skin, because of its dark red, œdematous lining which is curled up in folds. Two ureteral openings are present, and normal within the wall of the bladder.

On the right side just above the bladder, the ureter is thin walled and dilated so that it is about twice the size of the left ureter which is normal throughout its course. The dilatation of the right ureter is present for 5 centimeters above the bladder, and above this place it is normal. Below the place of dilatation of the right ureter which is the juxtavesical portion, this ureter is a contracted, rigid tube continuous through the bladder wall. The pelvis of each kidney, and the kidneys, are normal.

Histology. Serial sections were studied of the lower ends of the ureters from the abdominally exposed portions and upward for 3.8 centimeters, also serial sections of each ureter in its midportion. In the lower portion the wall consists almost entirely of epithelium of four to six cells thick, and loose mucous membrane in which small bundles of muscle fibers are enmeshed. These are predominately longitudinal fibers. Circular fibers are sparse. Chronic inflammation is extensive, in all portions

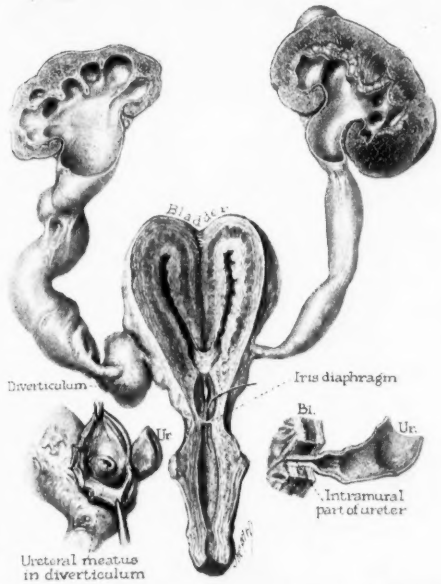

Fig. 8. Case 10. The right ureter empties into a diverticulum. Bladder wall enormously thickened. Iris type of valve in urethra. Intramural part of ureter thickened. Juxtavesical portion of left ureter thickened with dilatation above.

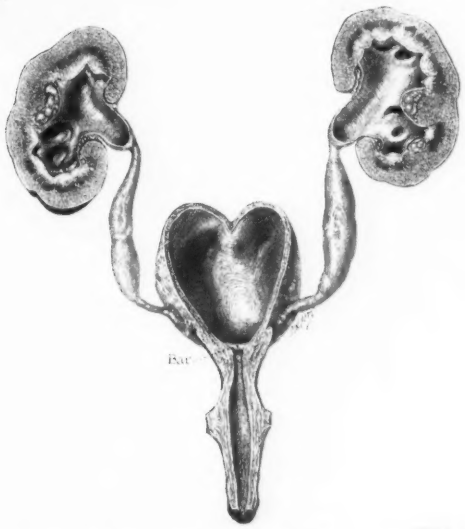

Fig. 9. Case 11. Note the dilatation of the bladder and presence of a median bar with dilatation of ureters above.

although most marked in the lining. Distally from the abdominal wall the findings are similar, except that the epithelium is less thick. In isolated places, however, the muscle fibers become prominent only to diminish quickly as the ureter is ascended. In the midportions of both ureters the walls are normally thin, the epithelium is narrow, the mucous membrane almost absent, and the thickness is largely that of the muscularis. The longitudinal fibers become less prominent so that near the kidneys the circular fibers predominate.

Case 6. F. L., female, aged 8 years, was admitted February 5, 1929, because of inability to walk, a sore throat, and marked irritability. The present complaints began a week ago with sore throat and fever. Three days before admission to the hospital the patient had a spell of vomiting, and 2 days later she was unable to walk. Recently she has been having some difficulty in swallowing. Examination revealed a well developed and well nourished female. The pharynx was moderately inflamed; no membrane and no paralysis. The heart, lungs, and abdomen were negative. Reflexes were as follows: knee-jerks, faint; Kernig's sign, negative; abdominal, present; Babinski, not elicited. Examination of the blood showed red cells 4,500,000; white cells 18,600; hæmoglobin 80 per cent. Blood chemistry showed non-protein nitrogen, 165; urea nitrogen, 145; and uric acid, 9. Blood pressure was systolic 90 and diastolic 80. Von Pirquet and Schick tests were negative. Examination of the urine showed albumin, pus, and a few casts. The patient developed a bronchopneumonia and died March 3, 1929. The clinical diagnosis was post-diphtheritic paralysis. Autopsy was done March 3, 1929. The anatomical diagnosis was chronic diffuse nephritis; bilateral hydroureters; chronic hyperplastic ureteritis; bronchopneumonia; caseous and calcified tuberculosis of lungs; parabronchial and mesenteric lymph glands.

Description of specimen (Fig. 4). There is a moderate amount of hydronephrosis, more marked on the right side. The right ureteropelvic junction is slightly narrower than the left. Both ureters show enormous dilatation. The walls are greatly thickened, the ureters are not tortuous, but resemble thick rigid tubes. The dilatation extends almost to the bladder wall.

Histology. Serial sections of the ureters were studied in ten different places and include all of the lower ureter. The sections of each ureter were essentially similar. In the intramural portion the epithelium and entire mucous membrane were swollen and of loose cellular structure. Two separate lymph nodes were present within the mucous membrane of the right ureter in its lower portion. In the bladder wall, the muscle layer of the ureter was retained as small bundles enmeshed in loose connective tissue.

Just above the intramural portions, the lining epithelium and the mucous membrane were similarly swollen, but the ureter thickness was greatly increased. This increase in size applied to the muscle bundles, about three-fourths of each bundle being

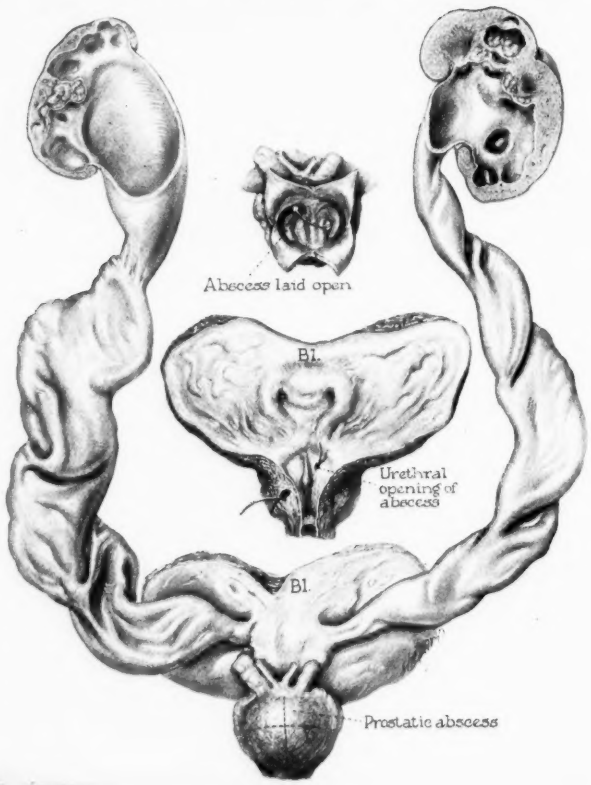

Fig. 10, left. Case 12. Uniformly dilated thin walled ureter. Intramural and juxtavesical part of the ureter thickened, due to muscle hypertrophy.

Fig. 11, right. Case 13. Note enormous thickening and dilatation of ureters beginning above the juxtavesical part. Probe passed through openings of prostatic abscess. Dotted line indicates incisions made in opening the prostatic abscess. Hydronephrosis, bilateral.

made up of longitudinal and one-fourth of circular fibers. The hypertrophy of muscle fibers was most marked in the peripheral portions of the sections. As the ureter was followed upward, the muscle hypertrophy persisted, but the predominance between longitudinal and circular fibers varied at different levels. All the muscle fibers stained well.

The lining of the ureter throughout its entire course was diffusely infiltrated with leucocytes in which the lymphocytes predominated. The inflammation was essentially of the mucous membrane.

Case 7. H. P., male, aged 7 months, admitted February 11, 1921; complaint, emaciation. Infant was a twin, full term, in a weakened condition at birth. Other infant has done very well, but this one failed to gain and emaciation has progressed since birth. Examination revealed an extremely emaciated child showing marked weakness. The skin was wrinkled and pasty grey in color. The head, neck, and heart were negative. There were distinctive signs of a disseminated bronchopneumonia. Emaciation of extremities was marked. Examination of the urine showed albumin and leucocytes. Death occurred on February 15, 1921, four days after admission. The clinical diagnosis was bronchopneumonia; atrophy, extreme. Autopsy was performed February 16, 1921. The anatomic diagnosis was bilateral bronchopneumonia; congenital stenosis of the left ureter; left hydroureter and hydronephrosis; ectopic right kidney.

Description of specimen. The right kidney is normal. The right ureter measures 78 millimeters from the bladder to the ureteropelvic junction. There is a fusiform dilatation 16 millimeters long and 6 millimeters wide in the lower one-half of the ureter. The ureteropelvic junction is normal. The ureteral orifices and the internal urethral orifice are normal. The ureter just above the bladder shows decided thickening. The left kidney contains fetal lobulations, and when opened reveals dilatation of the pelvis and a moderate hydronephrosis. The ureter measures 100 millimeters in length, and presents an unusual picture. At the ureterovesical junction it appears thickened for a distance of 7 millimeters. The next 45 millimeters are enormously dilated and the wall is unusually thin (Fig. 5). Above this point

of great dilatation the ureteral wall is enormously thickened, and midway between the previously described dilatation can be seen two transverse ridges. The pelvis is thin. There is no evidence of bladder neck obstruction.

Histology. Serial sections of the ureter were studied in eight grossly different places. These included the entire lower ureter on each side.

In the intramural and juxtavesical portions of the ureters the lumen is contracted, its lining being compressed into folds with an epithelial lining 3 to 5 cells thick. Here, the mucous membrane reticulum is abundant, but in the peripheral portions the longitudinal muscle fibers are at least twice the usual amount. The circular muscle fibers are normal. The fibers stain well. Except for one focal place where fibrous tissue is clumped inside the ureter wall, the mucous membrane is composed of loose connective tissue. Directly above the contracted juxtavesical portion the ureter balloons out, especially on the left side, and the wall is thin. The lining epithelium is narrow, the mucous membrane connective tissue almost absent, and the prominent muscle fibers are almost entirely circular. Above the dilated portion of the ureter the wall is again thick. The thickness consists of high epithelium, mucous membrane, and muscle fibers, circular and longitudinal in almost equal proportion.

CASE 8. D. F., female, aged 6 weeks, admitted August 18, 1927, because of vomiting of one week's duration and diarrhœa for 10 days. Vomiting, not projectile in character, occurred directly after feeding. There were four to five loose watery stools daily. No urinary symptoms. Examination revealed a very small, dehydrated baby. Physical examination was negative except for a moderate injection of the pharynx and a palpable liver. Examination of the blood showed red cells 3,600,000; white cells 15,400; hæmoglobin 62 per cent. A specimen of urine made postmortem showed many pus cells. Death occurred on August 20, 1927, *20 hours* after admission. The clinical diagnosis was gastro-enteritis. Autopsy was performed August 20, 1927. The anatomical diagnosis was bilateral duplication of the kidney pelves and ureters; pyo-ureter and pyonephrosis of upper half of the double right kidney; ectopic ureter (right upper) opening into the vagina.

Description of specimen (Fig. 6). On the left side the kidney is very much smaller than on the right and there is no evidence of hydronephrosis. There are two separate openings into the bladder. The right kidney shows two pelves and two ureters. The ureter from the lower half and the ureteral orifice are normal. The ureter that drains the upper half of the double kidney is markedly dilated, thin walled, and tortuous, and it runs under the bladder and urethra, terminating in the vagina. At the point of entrance of the ureter into the vagina there is a definite ring constriction. The opening of this aberrant or ectopic ureter is only 2 millimeters in circumference, at which point the ureter is dilated and measures 24 millimeters in circumference. There is no evidence

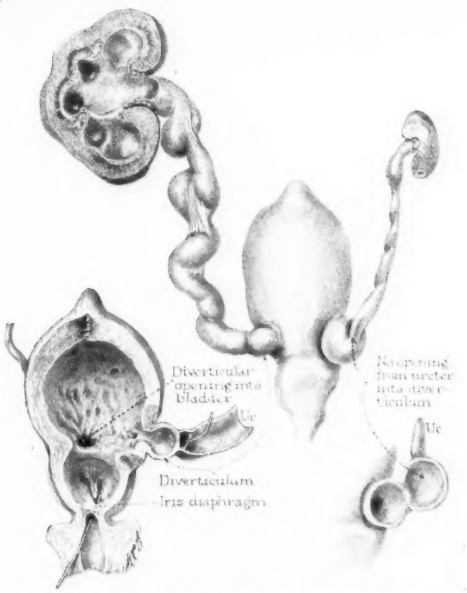

Fig. 12. Case 14. Note blind insertion of right ureter into a diverticulum. Aplasia of right kidney. The left ureter passes behind another diverticulitis. Iris valve in posterior urethra.

of obstruction in the bladder or urethra. The pelvis is exceedingly thin as is the rest of the ureter, and there is a marked hydronephrosis with compression-atrophy of the corresponding half of the kidney. There remains only a very thin shell of kidney tissue.

Histology. Of the ureter which comes from the upper one-half of the right kidney, serial sections are made of the entire lower portion upward from its opening into the vagina up to include its juxtavesical portion. Serial sections are also made of the midportion of the ureter. At the vagina, the lining of the ureter is in folds of thickly cellular epithelium, immediately beneath which the mucous membrane consists of dense, ramifying fibrous tissue which occupies about one-half of the thickness of the ureter. This fibrous tissue spreads outward and surrounds and compresses small bundles of longitudinal muscle. These muscles have undergone extensive hyaline degenerative changes. The fibrous tissue continues peripherally to the large and rather dense strands of circular muscle which is the essential peripheral layer of the ureter. These changes are present similarly through all sections up to and beyond the juxtavesical portion. In the midportion of the ureter the lining epithelium is similarly thick, and the mucous membrane is practically absent. Here the essential content of the wall is of smooth muscle, three-fourths of which is circular fibers. Throughout all sections

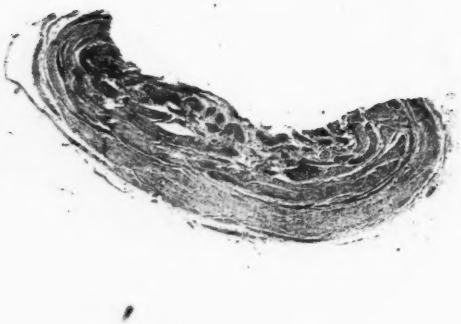

Fig. 13. Marked hypertrophy and hyperplasia of muscle bundles. (Mucous membrane removed.)

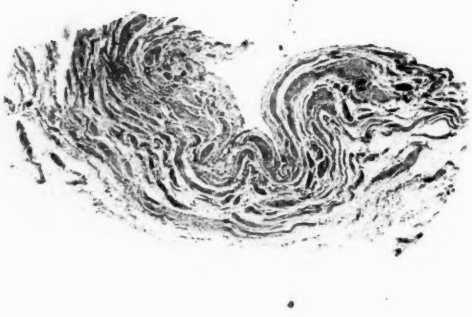

Fig. 14. Typical hypertrophy of the tunica muscularis (Mucous membrane removed.)

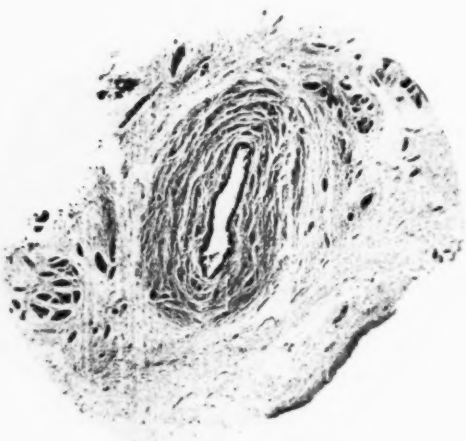

Fig. 15. Hypertrophy of longitudinal muscle fibers of the lower end of a ureter. (Hematoxylin and eosin stain.)

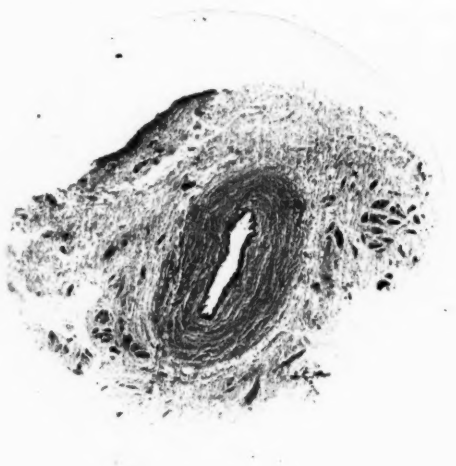

Fig. 16. Hypertrophy of muscle fibers of lower end of the ureter. (Van Gieson stain.)

from the vagina upward there is an infiltration of lymphocytes especially into the mucous membrane, and in places the lymphocytes are in clumps adjacent to muscle bundles. In the ureter, the changes are those of diffuse fibrosis, compression-atrophy of longitudinal muscle bundles, and chronic inflammation.

CASE 9. R. M., male, aged 20 months, admitted October 7, 1931, because of a mass in the abdomen and cramp-like pain at the beginning of urination. At 6 months of age the infant had pneumonia, and at 14 months, scarlet fever, during which time he passed *blood in the urine*. The mother stated that 1 month prior to admission she first noticed a "lump in the stomach," that it was larger at times and other times could not be felt. The right kidney was enlarged, irregular in outline, and easily palpable. In the midline was felt a suprapubic tumor which was

firm and movable and extended ½ inch below the umbilicus. On rectal examination, there was disclosed a non-fluctuant round mass about the size of a butternut, in the midline (thickened bladder). Roentgen-ray examination of the genito-urinary tract was negative for stone. Two attempts at intravenous pyelography with skiodan failed to visualize the urinary tract. The passage of a soft rubber catheter met an obstruction in the prostatic urethra. The obstruction was finally overcome and 180 cubic centimeters of urine were obtained. Examination of the urine showed albumin, 3+; pus, 4+; no red cells or casts. Culture of the bladder urine revealed streptococci and Staphylococcus aureus. A cystogram made at this time showed a bilateral hydronephrosis. Examination of the blood showed red cells, 3,800,000; white cells, 8,400; hæmoglogin, 65 per cent; neutrophiles, 56 per cent; and lymphocytes, 44 per cent. Blood chemistry: non protein nitrogen, 112; creatinin, 2; and uric acid, 6.5. The infant ran a septic course. Death occurred on October 22, 1931. The clinical diagnosis was congenital anomaly of the urinary tract (probably a vesical neck obstruction or valve), with secondary bilateral hydroureters and uræmia. An autopsy was performed October 23, 1931. The anatomical diagnosis was congenital valve obstruction of the posterior urethra; median bar obstruction; dilatation and hypertrophy of the bladder; diverticulum of the bladder; bilateral hydroureter and hydronephrosis; pressure atrophy of kidneys.

Description of specimen (Fig. 7). The right kidney is enlarged, its surface is irregular, the cortex thin, the pelvis extremely dilated, marked hydronephrosis. Similar changes were noted in the left kidney, but it is smaller than the right. Both ureters are markedly dilated and the walls are thin. The dilatation extends from the renal pelvis down to the bladder wall. The intramural part of the ureters is not dilated. Great thickening of the bladder wall is noted. An opening of a diverticulum is seen in the upper right quadrant. There is hypertrophy of the trigone, and the ureteral openings are normal. The presence of a median bar obstruction is seen. The prostatic urethra is dilated, and in front of it is seen a fold of the mucous membrane.

Histology. Serial sections were made of the entire lower portion and the midportion of each ureter. The findings of each ureter are essentially similar. In the intramural portion the lining epithelium is narrow, the mucous membrane normally thick, and the inner circular and outer longitudinal muscle fibers are only normally prominent. Just above the bladder, however, the diameter of the ureter and the thickness of the wall are markedly increased. The lining epithelium and mucous membrane are narrow. The thickness of the wall is due to a marked increase in the size of the muscle fibers in the large bundles held together by loose connective tissue. At least three-fourths of the thickness of the ureteral wall is muscle tissue. Here, in places, the increase of muscle is due to longitudinal fibers in clumps, and on opposite sides of the same block the circular muscle fibers are densely prominent. This variability of muscle prominence is evident throughout all the sections of the lower portions of the ureters. In the midportion of each ureter there is a uniform hyperplasia of all layers, and not of muscle fibers only. Here the longitudinal fibers are central, the circular fibers peripheral. In all sections of the ureter there is evidence of chronic inflammation. This is most marked in the mucous membrane.

CASE 10. R. K., male, aged 2 months, admitted October 5, 1931, because of frequent projectile vomiting, occurring 5 to 10 minutes after feeding. This symptom has been present since the child was 12 days old. A hard swelling was noted in the lower abdomen which the mother first noticed 3 weeks ago; also swelling of the left leg for 2 weeks. The mother stated that the infant urinated very infrequently. Examination revealed a somewhat underweight and mildly dehydrated infant. The abdomen was distended and tense, with dilated superficial veins. A suprapubic tumor, extending 2.5 centimeters above the umbilicus, was felt in the lower abdomen. The right kidney was palpated but not the left. There was some œdema of the left leg. The cystogram showed a large, slightly asymmetrical bladder and a markedly dilated left ureter. The right ureter was not visualized. The intravenous pyelogram failed to show any evidence of dye in the kidney regions or along the course of the ureters. Examination of the blood showed red cells, 3,800,000; white cells, 18,500; hæmoglobin, 55 per cent. Blood chemistry: non-protein nitrogen, 42; uric acid, 5. Examination of the urine showed albumin, many pus cells, and no casts. The infant was treated by means of indwelling catheter, but ran a progressively unsatisfactory course, growing more and more dehydrated in spite of the fluids given parenterally. Death occurred on December 31, 1931. The clinical diagnosis was vesical neck obstruction; bilateral hydroureter with hydronephrosis; uræmia; bronchopneumonia. Autopsy was done January 1, 1932. The anatomical diagnosis was congenital iris valve of the posterior urethra; dilatation and hypertrophy of the bladder with *diverticulum* formation; bilateral hydronephrosis; atrophy of the left kidney; bilateral pyelonephritis; hypostatic bronchopneumonia.

Description of specimen (Fig. 8). In front of the verumontanum is seen the presence of an iris diaphragm. It completely closes the urethra except for a pinpoint opening, and the prostatic urethra is somewhat dilated. The bladder wall is enormously hypertrophied and there is the opening of a diverticulum at the site of the normal insertion of the right ureter. The right ureter opens into this diverticulum, and the right ureteral orifice is normal. Immediately above this diverticulum the ureter is thickened but of normal circumference, but above this point it is enormously dilated, twisted, and sacculated. The pelvis is dilated and the kidney shows hydronephrotic atrophy, with only a small shell of the kidney re-

maining. The pelvis is 48 millimeters in circumference. The left kidney reveals a cortical abscess and moderate hydronephrosis. It measures 44 by 30 by 26 millimeters. The pelvis measures 20 millimeters in circumference and 27 millimeters in length. The lower ureter is thin walled, tortuous, and dilated. At its entrance into the bladder it is thickened and narrowed.

Histology. Serial sections are made of the entire lower end of each ureter. In the left ureter in the intramural portion the lining epithelium is thick, high, and compressed into folds. The mucous membrane is wide and its connective tissue extends peripherally so as to surround small circular muscle fibers, adjacent to which are masses of longitudinal muscle fibers of about uniform size, with poor staining properties. Directly above the bladder, the ureter wall is thick. Its lining epithelium is narrow, the mucous membrane wide. Here the muscle fibers are prominent and are circular and longitudinal in almost equal proportions. The longitudinal fibers are surrounded by dense connective tissue and in a few places some of them stain poorly, similar to what occurs in atrophy. Immediately adjacent, however, the muscle bundles are markedly hypertrophic and some of them are two to three times the usual size. This hypertrophy continues upward to the portion where the dilatation begins. On the right side, at the point of entrance of the ureter into the diverticulum, the lining epithelium is thick. Here there is a definite increase of fibrous tissue throughout the mucous membrane, and in places it compresses or replaces muscle fibers as in stricture. Directly above this part of the ureter, and extending to the dilated part the muscle fibers, especially the peripheral longitudinal ones, are hypertrophic in similar fashion to those of the left ureter. Along the entire course of the ureter and into the kidney proper there are rather extensive changes of acute inflammation.

CASE 11. J. D., male, aged 5 months, admitted January 24, 1932, because of the following complaints: Stiff neck of 3 days' duration, pneumonia for 11 days, fever of 102 to 106 degrees for 18 days, drowsiness, cough, anorexia for 2 days, dyspnœa for 1 week, and stiffness of the right arm. There were no urinary symptoms.

Examination revealed a well nourished, acutely ill infant, with labored and irregular respirations. The neck was rigid. There was bulging of both eardrums, an area of dullness over the right chest, anteriorly and posteriorly, and bronchial breathing over the entire chest, most marked over the upper lobe. There were a few coarse râles. The heart was normal except for sinus arrhythmia. The abdomen was negative except for considerable distention. The spinal fluid showed Bacillus influenzæ in pure culture. Death occurred 10 hours after admission. The clinical diagnosis was bronchopneumonia; meningitis (influenzal); otitis media. An autopsy was done January 26, 1932. The anatomic diagnosis was fibropurulent cerebrospinal leptomeningitis (Bacillus influenzæ); cavernous sinus thrombophlebitis; bilateral purulent otitis media; bilateral bronchopneumonia; fibrino-purulent right pleuritis (Bacillus influenzæ); serous peritonitis (Bacillus influenzæ); median bar formation; bilateral hydronephrosis.

Description of specimen (Fig. 9). The bladder is dilated, the wall is somewhat thickened uniformly, and the presence of a median bar is noted. The ureteral orifices are normal. The lower ends of the ureters are contracted and the lumen is small. The right ureter, at a point 15 millimeters above the bladder, is dilated, the dilatation continues upward to, or nearly to, the ureteropelvic junction. The left ureter shows dilatation at a point about 5 millimeters above the bladder, and this dilatation extends to the ureteropelvic junction. The intramural part of the ureter appears to be of about the same size and structure as the juxtavesical part. The kidneys show moderate hydronephrosis.

Histology. In serial sections of both ureters, the findings are essentially similar. In the ureters within the bladder wall the lining epithelium and the mucous membrane are normally wide, the muscle fibers scant. At the point where the right ureter enters the bladder the mucous membrane is thick and in places ramified by strands of fibrous tissue which surrounds small bundles of centrally placed longitudinal muscle fibers. As the ureter is ascended, however, the central longitudinal and peripheral circular muscle followed upward are markedly increased in size, and they occupy about three-fifths of the entire ureteral wall. In the left ureter, from the bladder junction up to the dilated portion of the ureter, the muscle fibers are generally huge. The longitudinal fibers are centrally placed, the circular fibers peripheral. This hypertrophy of muscle is of both types, and may be present in a single section, or in zones as the ureter is ascended, where one type predominates over the other. In the upper two-thirds of each ureter the walls are thin. Here the epithelium is narrow, the mucous membrane rather diffusely fibrous, and the muscle tissue scanty.

CASE 12. R. O., male, aged 6 weeks, admitted December 9, 1927, because of vomiting. At the tenth to twelfth day of life he began to vomit once daily, the vomiting being projectile in type. The frequency of the vomiting gradually increased to three to four times daily after each feeding. He began to cough 10 days ago. For the past 2 days breathing has been very rapid. Temperature upon admission was 97 degrees. Examination revealed a dehydrated and malnourished child, breathing deeply and very rapidly; no cyanosis. The lungs were negative. There was a systolic murmur at the precordium and apex. The abdomen showed no palpable masses. The extremities were negative. The infant was too ill for a thorough examination.

Examination of the blood showed red cells, 2,700,000; white cells, 16,900; hæmoglobin, 36 per cent; neutrophiles, 73 per cent; lymphocytes, 27 per cent. No examination of urine was made. The following day there was a change for the worse; he vomited dark brown material and a small amount of

mucus. Respirations were rapid and gasping, and there were many râles throughout the chest. Pulmonary œdema was noted. Death occurred *30 hours* after admission. The clinical diagnosis was pylorospasm; pulmonary œdema. An autopsy was done December 11, 1927. The anatomical diagnosis was marked bilateral hydroureters and hydronephrosis with infection; extensive bilateral bronchopneumonia; emaciation; dehydration; generalized anæmia.

Description of specimen (Fig. 10). The bladder is dilated half way to the navel. There is marked dilatation of the ureters which are thin walled and have a tortuous course. The pelves are dilated, with marked destruction of the kidney substance. There is marked hydronephrosis. Both ureters are dilated down to the juxtavesical part, at which point the walls of the ureter are extremely thick. The bladder wall is also thickened. Both ureteral orifices are normal. There is no obstruction in the urethra.

Histology. In serial sections of the right ureter in its lower portion, at, and directly above, the bladder the lining epithelium is thin. In large circular strands and in large longitudinal bundles, the muscle fibers completely surround the lumen and occupy also most of the mucous membrane layer. A few of the large fibers are hyalinized and some have vacuoles in them where the connective tissue is more evident. This hypertrophy is perhaps a little more marked in the circular muscle fibers. In places, large muscle fibers are present, extending from the lining epithelium to the peripheral connective tissue. The entire ureter from the juxtavesical portion to the kidney pelvis has a thin wall, composed of all layers, but the mucous membrane is almost completely obliterated. Here all the tissues stain poorly.

CASE 13. W. K., male, aged 9½ years, admitted March 7, 1930, because of a swelling of the right side of the scrotum. The swelling had been present for 2 weeks and had reached the size of a hen's egg. It ruptured and drained for several days, then healed over, and remained painful and larger than normal. A similar condition had occurred in the left side of the scrotum a year previously and had drained for 4 weeks. The left testicle was removed and a diagnosis of tuberculosis was made. For a month before admission to the hospital he had lost weight and his appetite was poor. Since he was 5 months old he has had a skin affection, first on the buttocks, afterward becoming gradually generalized. The urinary stream was very fine and there was obstruction to urination. Examination revealed an underdeveloped and poorly nourished child. There was a mucoid discharge from the ears. The teeth were carious and loose. The tonsils were large. The heart and lungs were negative. The bladder was distended as high as the umbilicus. Pus was oozing from the urethral meatus. The left testicle was absent and there was a small healed sinus on the left side of the scrotum. The right testicle was large, hard, and painful. There was an atrophic condition of the skin of the face, and of the extremities and abdomen as far as the umbilicus. Roentgen-ray examination revealed a sacral spina bifida occulta. An intravenous pyelogram was attempted but was unsuccessful on account of renal insufficiency. Examination of the blood revealed red cells, 3,450,000; white cells, 10,800; hæmoglobin, 30 per cent. Examination of the urine showed a small amount of albumin and an excessive amount of pus cells. Tubercle bacilli not noted. Various tuberculin tests were negative. Blood chemistry: non-protein nitrogen, 84; uric acid, 8.6; and creatinin, 2. An abscess in the right scrotal region was incised, the pus of which contained hæmolytic streptococci in pure culture. An indwelling catheter was inserted and operation advised, but he ran a septic temperature, and became less clear mentally. Death occurred on April 29, 1930. The clinical diagnosis was chronic urinary retention with renal insufficiency and infection; obstruction of the posterior urethra; acute epididymitis; infantilism and xeroderma. An autopsy was done April 29, 1930. The anatomical diagnosis was prostatic abscess producing obstruction of the posterior urethra; purulent cystitis; hypertrophy of the bladder wall; bilateral pyonephrosis and pyelonephritis; septicæmia (streptococcus hæmolyticus); bronchopneumonia; partial prolapse of the rectum; dwarfism.

Description of the specimen (Fig. 11). The bladder wall is enormously thickened and hypertrophied. There is no evidence of urethral valve formation or strictures. The ureteral openings themselves are normal. On the floor of the prostatic urethra, on the left side, is an opening that measures 2 millimeters in diameter. A smaller opening measuring 3 millimeters is located outside the lining of the mucous membrane of the urethra. A probe inserted in one opening reveals a large cavity in the prostate, and the two previously described openings communicate. The cause for the enormous hypertrophy and thickening of the bladder wall is evidently due to an abscess of the prostate that ruptured into the prostatic urethra, filled up with urine, and caused a chronic obstruction. Examination of the kidneys shows marked hydronephrotic atrophy. Very little kidney tissue remains. The pelvis of the right kidney measures 45 millimeters across, and from the edge of the kidney to the ureteropelvic junction, 60 millimeters. Below this point the ureter is enormously dilated, tortuous, falls on itself, and measures 80 millimeters in its largest circumference. As the ureter is followed down it gradually narrows to the juxtavesical portion of the ureter, where it is thickened and hard, and stands out in marked contrast to the thick walled, dilated portions of the ureter above. The circumference of the ureter here is only 16 millimeters.

The left kidney is 62 millimeters long, 37 millimeters wide, and 20 millimeters thick. The pelvis is much smaller and measures in its widest part 50 millimeters in circumference, and the length down to the ureteropelvic junction is 35 millimeters. The widest part of the left ureteral wall is not as wide as the right wall, its circumference being 90 millimeters.

The ureter is dilated down to its bladder end, and as the intramural part is dissected out, the wall of the ureter is thick.

Histology. Serial sections are made of each ureter in the intramural and juxtavesical portions, also of each ureter in its midportion. The changes in each ureter are essentially similar. In the bladder wall and directly above it, the lining epithelium and mucous membrane are thin and are the site of extensive subacute and chronic inflammation. Compressing and partially obliterating the mucous membrane are huge masses of muscle fibers which completely surround the ureter. Here the predominant muscle is circular, and all fibers stain well. The hypertrophy of muscle tissue is of the entire juxtavesical portions of the ureters up to the point where dilatation begins.

In the midportion, the ureter wall is thick, this being due largely to chronic inflammatory hyperplasia. Here the muscle fibers are peripherally located and are about normal size in proportion to the thickness of the wall. These portions stain poorly.

CASE 14. A. N., male, aged 12 days, was admitted March 12, 1932. For the first 60 hours following birth, no urine was passed. Four days before admission to the hospital the amount of urine passed was very small and the voiding was attended with great difficulty. Vomiting persisted for 4 days. Examination revealed a dehydrated infant with poor tissue turgor. There was a purulent nasal discharge. The tongue dry and heavily coated. There were a few small pustules on the right side of chest. Rhonchi heard in all lung areas. A remnant of the umbilical cord was attached. The bladder was distended up to one fingersbreadth above the navel. Following the catheterization the infant voided frequently small amounts. However, a suprapubic tumor rapidly developed. The size of the bladder greatly increased until it reached nearly to the navel. Roentgen-ray examination was negative for stone. An intravenous pyelogram was made—no visualization on the right side. On the left side the urogram showed a hydronephrosis and hydroureter. The cystogram showed an enlarged bladder.

Examination of the blood revealed red cells, 6,500,000; white cells, 4,700; neutrophiles, 37 per cent; and lymphocytes, 58. Voided specimens of urine contained albumin, many red blood cells, no casts; loaded with white blood cells. Von Pirquet tests were negative. Staphylococcus aureus in pure culture were obtained from the blood. On March 17, the infant began to vomit and an impetigo began to spread. The next day the impetigo became much worse, spreading rapidly and appeared as a diffuse progressing superficial inflammation from which the skin exfoliated in sheets without the formation of bullæ. Almost the entire body became involved and this was followed quickly by death on March 20. The clinical diagnosis was congenital bladder neck obstruction; left hydronephrosis and hydroureter; exfoliative dermatitis neonatorum (Ritter's disease). An autopsy was done March 21, 1932. The anatomical diagnosis was congenital iris-diaphragm obstruction of the prostatic portion of the urethra; huge hypertrophy and dilatation of the urinary bladder with diverticulum formation; left hydroureter and hydronephrosis; acute suppurative urethritis, cystitis, and pyelonephritis, (Staphylococcus aureus hæmolyticus, Streptococcus hæmolyticus); congenital atresia of the right ureter; aplasia of the right kidney; uræmia; hypostatic bronchopneumonia; dehydration; emaciation; excoriation of the skin of the perineum; dermatitis exfoliativa neonatorium (impetigo); Meckle's diverticulum.

Description of specimen (Fig. 12). The bladder wall is very thick. Near the normal location of the right ureteral orifice is seen the opening of a diverticulum. The right ureter fuses with the diverticulum on the right side. The lower end of the right ureter is closed. Both right ureter and kidney show marked aplasia. The left ureteral orifice is normal. In front of it is seen the opening of another diverticulum. The intramural portion of the ureter is thickened and above the thickening the ureter is dilated and tortuous. There is a marked left hydronephrosis. An iris type of valve formation is noted in the posterior urethra.

Histology. In serial sections of the right ureter which has a blind attachment to a diverticulum of the bladder, from its lower portion to the kidney, the lumen is patent. The ureter wall has a normally thick epithelium, but all of the remainder of the wall is a loose fibrous reticulum extending to the periphery. In this reticulum are small bundles of longitudinal muscle fibers and solitary fibers of circular muscle. In the kidney there is extensive fibrosis, atrophy of the tubules, and many hyalinized glomeruli. In one place of the cortex, cartilage is present. The wall of the diverticulum of the bladder at the lower end of the right ureter has a flat epithelial lining of low cuboid cells. The mucous membrane adjacent is richly vascular, and has changes of subacute inflammation. Here the mucous membrane is thick, occupies about two-thirds of the diverticulum wall, and in the periphery are delicate strands of muscle fibers which stain poorly. In serial sections of the entire lower part of the left ureter throughout the entire portion, compressed by a diverticulum of the bladder on this side, the increase in size of the ureter wall is largely due to a huge hypertrophy of muscle fibers. Here the predominant muscle fibers are circular, and are mainly peripheral. In places, three-fourths of the thickness of the wall of the ureter consist of muscle fibers which surround the lumen. At those points where the muscle fibers are less dense the thickness consists of mucous membrane.

In serial sections of the midportion of the left ureter, the lining epithelium and mucous membrane are narrow. The muscle fibers are scant, and consist essentially of circular fibers. The wall is thin in comparison with the juxtavesical portion.

CASE 15. W. L., male, aged 6 weeks, admitted July 14, 1932, because of a history of diarrhœa, fever

of 24 hours' duration, and anorexia for 2 weeks. He had been a feeding problem since birth. Examination revealed a markedly anæmic, dehydrated, emaciated, and cyanotic infant, with rapid and shallow respirations. The head was oddly shaped, the parietal and occipital bones bulging. The fontanels and cranial sutures were widely separated, and here the soft tissues were depressed. The abdomen was distended, tight, and bulging at the flanks. There were physical findings of a diffuse bronchopneumonia.

Examination of the blood showed red cells, 2,250,000; white cells, 51,400; hæmoglobin, 60 per cent; polymorphonuclears, 65 per cent; large lymphocytes, 32 per cent; mononuclears, 3 per cent. No urine was obtained, although a test tube was fastened to the penis. The spinal fluid was under normal pressure, 7 cells per cubic millimeter, and negative to bacteriological culture and Wassermann and Kahn reactions. There was occult blood in the stools. Death occurred 31 hours after admission. The clinical diagnosis was intestinal intoxication; bronchopneumonia; decomposition; Mongolinism. An autopsy was done July 16, 1932. The anatomical diagnosis was congenital membranous valve obstruction of the prostatic portion of the urethra; hypertrophy and dilatation of the urinary bladder; hydroureter and hydronephrosis; marked compression atrophy of the kidney substance; extensive fibrinopurulent cystitis, ureteritis, and pyelonephritis (Bacillus coli communis); acute hyperæmia of the nasopharynx; extensive bilateral bronchopneumonia; hyperplasia of the peribronchial lymph nodes and of the conglomerate lymphoid tissue of the lining of the ileum; hyperplasia of the mesenteric lymph nodes; paralytic ileus; generalized anæmia; Mongolian idiocy; dehydration; emaciation; widely-separated sutures of the cranial bones; phimosis; multiple lumbar spinal needle puncture wounds; multiple subcutaneous hypodermoclysis needle puncture wounds.

Description of specimen. The kidneys, ureters, urinary bladder, and entire urethra are included in one mass and are carefully dissected. The entire urethra is incised from the glans penis back into the bladder and continued up in front of the bladder to its back portion. The lining of the urethra is normally smooth throughout its entire course, but at the beginning of the prostatic portion of the urethra there is a high linear fold which extends back to the front margin of the verumontanum. In general, this congenital membranous fold of the prostatic urethra is "V" shaped, the apex of the "V" pointing toward the verumontanum. The walls of the fold are thin and easily compressible. It is assumed that urine within the bladder would cause an obstruction at the membranous portion of the prostatic urethra by displacing the fold either to the right or left thus making a pocket, and the "V" shape of the fold would similarly cause difficulty of catheterization from below when the fold was displaced down and laterally by a distended bladder. The fold of the prostatic portion of the urethra occupies almost the entire caliber of the urethra at this point. The verumontanum is about twice its usual size, and the urinary bladder wall adjacent is thickened, as is that of the body of the urinary bladder generally. The lining of the urinary bladder is coarsely trabeculated. The intramural portions of each ureter are narrow, and directly above the bladder, each ureter, uniformly for a distance of 20 millimeters, is markedly contracted into a narrow firm tube. The right ureter in this juxtavesical portion is cord-like. Both ureters, 20 millimeters above the bladder, are markedly and uniformly dilated and slightly sacculated throughout their entire length. The pelves of both kidneys are dilated so that they are at least 3 to 4 times the usual size. Hydronephrosis is more marked on the left side. In surfaces made by cutting the kidneys there is hydronephrotic atrophy so that the cortex is in most places only 5 to 7 millimeters thick. At least one-third of the kidney substance has been destroyed. The content of the kidney pelves, ureters, and bladder consists of purulent urine.

Histology. In serial sections of the entire lower one-third of each ureter from the intramural portions upward, the changes of each ureter are essentially identical. Within the bladder wall, the epithelial lining is narrow, the connective tissue of the mucous membrane is normally thick and homogeneous. Here longitudinal muscle bundles are present and some of them have changes of atrophy. Circular muscle fibers are scant. In the peripheral portions of the ureter here, the longitudinal muscle fibers are huge and extend laterally to blend with those of the bladder wall. Adjacent to the bladder wall, and for 20 millimeters upward, the mucous membrane becomes narrow and within it and peripherally to it, the longitudinal fibers are so abundant as to occupy about three-fourths of the thickness of the wall. Strands of circular muscle fibers ramify around these longitudinal bundles. Above this juxtavesical portion, the epithelium is more abundant, the mucous membrane connective tissue is less compact, and as the ureter is traced upward, the narrow, densely muscular juxtavesical portion gradually dilates. Here the circular muscle fibers are the predominant muscle. At about the middle third of the ureter and up to the kidney the wall is thin. This atrophy is of all layers but especially of the mucous membrane. From the bladder upward to the kidneys on both sides there is subacute inflammation which is present throughout the ureter but especially in the mucous membrane.

In reviewing this series of cases we note that 12 patients were males and 3 were females. The youngest child was 1 month and the oldest 10 years, and 9 were under *7 months* of age. The clinical diagnoses were exceedingly interesting and showed, in a large majority, that the urological condition was not recognized. This was in part due to

the fact that some of the patients were seen in the pre-urological days of pediatrics and also due to the fact that some of the patients entered the hospital practically *in extremis* so that a complete study was not possible. On the other hand, it is exceedingly interesting to note the accuracy of the urological diagnosis in the recent cases since the establishment of the urological service, since which time the attention of the general services has been directed to the possibility of these various lesions so that the diagnoses were made before the cases were referred to the urological service.

A detailed summary of the ages, sex, clinical diagnoses, with a brief résumé of the autopsy findings is given in Table I.

An analysis of this group shows that the underlying pathology was essentially obstructive in nature and that in most of the cases the obstruction was congenital in origin. In this group were found the presence of congenital valves in the posterior urethra, the presence of median bars, the presence of diverticula with the ureters inserted into the diverticulum.

The presence of obstruction soon leads to urinary stasis and dilatation, and these patients are prone to urinary infections. The infections may follow the onset of an acute infectious process elsewhere in the body, as, for example, an acute upper respiratory infection, diarrhœa, or one of the acute infectious diseases such as measles; or infection may follow the passage of sounds, catheters, and cystoscopes.

HISTOLOGY

Histological studies were carried out to coronal serial sections of the entire intramural and juxtavesical portions of the ureter as well as all other parts of the ureter that showed gross abnormality. Hæmotoxylin and eosin, Van Gieson, and Weigert stains were used.

As a result of these studies, we found that the obstruction in the vesical end of the ureter in 12 cases was due predominantly to hypertrophy of muscle, most of which was of the longitudinal fibers. For purposes of comparison a series of normal ureters was removed at autopsy and similar serial sections were made.

In 3 of the cases, instead of muscle hypertrophy, the lower ends of the ureter showed changes compatible with fibrous or true stricture. In this group there was a predominance of scar tissue which replaced the normal muscle to a very large degree, so that we had no difficulty in differentiating between these two types of lesions. In these cases of true fibrous stricture we found an anomalous insertion of the ureter in 3 cases; in 1, a double kidney, one ureter terminating in the vagina; in 2 cases the ureter terminated in a diverticulum; in 1 the end of the ureter that was inserted in the diverticulum was completely closed by scar formation.

In seeking an explanation for the presence of the hydronephrosis we found that these cases fall into 3 groups.

1. In the first group the hydronephroses were due to the presence of a true fibrous stricture of the ureter, and here we note the presence of an anomalous insertion of the ureter in the 3 cases. In 1 case the accessory ureter with fibrous stricture was inserted in the vagina. In the 2 remaining cases the ureter terminated in a diverticulum.

2. In 11 cases of hydronephrosis the obstruction in the ureter was due to extensive hypertrophy of the muscular coat of the ureter and this involved chiefly the longitudinal fibers. It is exceedingly interesting to note that this hypertrophy was associated with obstructive lesions at or in front of the vesical orifice, such as median bars (3 cases), the presence of congenital valves in the urethra (6 cases), and in 1 case, due to an abscess in the prostate that produced obstruction to urination (Case 13). It would appear, therefore, that the muscular hypertrophy was directly associated with the presence of the obstruction. The explanation for the hypertrophy can be readily explained on an anatomical basis.

In 1812, Sir Charles Bell called attention to the fact that some of the muscle bundles of the ureter descend from the orifices of the ureter toward the orifice of the bladder. Here the fibers from each side unite and run toward the prostate. The longitudinal fibers

TABLE I.—SUMMARY OF CASES REPORTED

Case No. Sex	Age	Clinical diagnosis	Urological conditions	Cause of death	Autopsy findings
1 M	1 mo.	Pyloric obstruction	Not recognized	Broncho-pneumonia	Congenital valve in posterior urethra; hypertrophy and dilatation of urinary bladder; hydroureter and hydronephrosis, bilateral
2 M	1 yr.	Spina bifida; fractured femur; talipes equinovalgus	Not recognized	Broncho-pneumonia	Spina bifida; horseshoe-shape kidney; hydroureters and hydronephrosis
3 M	11 mos.	Alimentary intoxication	Not recognized	Alimentary intoxication	Ectopic right kidney; hydroureter and hydronephrosis, diverticulum of bladder, left; emaciation and anæmia
4 M	4 yrs.	Acute pyelocystitis	Not recognized	Septicæmia	Congenital valve in posterior urethra; median bar; hypertrophy of bladder wall; chronic cystitis; bilateral hydronephrosis; dilatation of ureters; multiple abscesses of kidneys
5 F	5½ mos.	Exstrophy of bladder; acute pyelonephritis	Not recognized	Broncho-pneumonia	Exstrophy of bladder; subacute cystitis and urethritis; empyema; ventral hernia
6 F	8½ yrs.	Uræmia; pyelonephritis; postdiphtheritic	Not recognized	Uræmia	Bilateral hydroureters; chronic ureteritis; chronic diffuse nephritis; caseous and calcified tuberculosis of lungs; parabronchial and mesenteric lymph glands
7 M	6½ mos.	Bronchopneumonia; extreme atrophy	Not recognized	Bronchopneumonia; extreme atrophy	Ectopic right kidney; congenital stenosis of left ureter; hydroureter and hydronephrosis
8 F	1½ mos.	Gastro-enteritis	Not recognized	Gastro-enteritis	Ectopic right ureter ending in vagina; bilateral duplication of kidney pelves and ureters; pyo-ureter and pyonephrosis of upper half of double right kidney
9 M	1¼ yrs.	Congenital anomaly of urinary tract—probably vesical neck obstruction or valve—with hydroureters and uræmia	Recognized	Uræmia	Congenital valve of posterior urethra; median bar; dilatation and hypertrophy of bladder; diverticulum of bladder; bilateral hydroureter and hydronephrosis
10 M	3 mos.	Vesical neck obstruction with hydroureter and hydronephrosis	Recognized	Broncho-pneumonia	Congenital iris valve of posterior urethra; bladder diverticulum; bilateral hydronephrosis; atrophy of left kidney; bilateral pyelonephritis; hypostatic bronchopneumonia
11 M	5 mos.	Bronchopneumonia; meningitis	Not recognized	Bronchopneumonia; meningitis (B. influenza)	Median bar formation; bilateral hydronephrosis; fibropurulent cerebrospinal leptomeningitis; cavernous sinus thrombophlebitis; bilateral purulent otitis media bilateral bronchopneumonia; fibrinopurulent right pleuritis; serous peritonitis
12 M	6 wks.	Pylorospasm; pulmonary œdema	Not recognized	Broncho-pneumonia	Marked bilateral hydroureters and hydronephrosis with infection; extensive bilateral bronchopneumonia; emaciation; dehydration
13 M	10 yrs.	Obstruction of posterior urethra; chronic urinary retention with renal insufficiency and infection	Recognized	Septicæmia	Prostatic abscess producing obstruction of posterior urethra; purulent cystitis; hypertrophy of bladder wall; bilateral pyonephrosis and pyelonephritis; septicæmia (streptococcus hæmolyticus); bronchopneumonia; partial prolapse of the rectum
14 M	12 days	Congenital bladder neck obstruction; left hydronephrosis and hydroureter; exfoliative dermatitis	Recognized	Septicæmia	Congenital iris-diaphragm of prostatic urethra; hypertrophy and dilatation of urinary bladder with diverticulum formation; left hydroureter and hydronephrosis; acute suppurative urethritis and pyelonephritis; congenital atresia of right ureter; aplasia of right kidney
15 M	2 mos.	Intestinal intoxication; bronchopneumonia	Not recognized	Broncho-pneumonia	Congenital valve of prostatic urethra; marked hydroureter and hydronephrosis; fibrinopurulent cystitis; ureteritis and pyelonephritis; bilateral bronchopneumonia; paralytic ileus

are also known as Bell's muscles. Some of the ureteral fibers pass medially to form the interureteric ligament or Mercier's bar. These findings were verified by Ellis in 1858.

As is well known, in certain obstructions, for example in benign hypertrophy of the prostate, it is not uncommon to find hypertrophy of the trigone. Wesson has recently studied the musculature of the trigone. According to his studies, the trigonal muscle is a definite entity arising from the longitudinal muscle bundles of the ureters and is superimposed on the muscles of the bladder wall. Wesson furthermore stated that in his opinion the trigonal muscle plays an active part in the process of micturition, as it pulls open mechanically the internal vesical orifice.

From these anatomical facts we believe that the hypertrophy of the muscle bundles found in the lower end of the ureters in this group of cases was the hypertrophy of overwork in an attempt to overcome the obstruction at or in front of the bladder.

In one case of muscle hypertrophy the autopsy records fail to state whether or not obstruction was present at or in front of the bladder orifice, but this we believe was present. Only the part of the bladder wall around the ureter was present in the museum specimen.

3. In the 2 remaining cases no mechanical obstruction was noted; namely, in the case of ectopia of the bladder and in the case of extensive postdiphtheritic paralysis. The muscle hypertrophy here was probably due to disturbed physiology in the one case (ectopic bladder) and due to disturbed physiology secondary to disturbance in the nervous mechanism following the diphtheritic paralysis.

SUMMARY

1. Stricture of the ureter, in the generally accepted terms, producing hydroureter and hydronephrosis, was present in only 3 cases all of whom had an anomalous insertion of the ureter.

2. In the 12 remaining cases, the obstruction was due to hypertrophy of the muscular coat of the ureter (longitudinal fibers).

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SPINAL ANÆSTHESIA

A Clinical and Experimental Study

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PERHAPS no subject in surgery has aroused more interest in recent years than that of spinal (subarachnoid) anæsthesia. The remarkable relaxation, the absence of interference by large distended loops of bowel, and the smooth postoperative course have greatly facilitated surgical procedures especially within the abdomen and have made the surgeon accustomed to its use unwilling to return to other forms of anæsthesia. On the other hand, the almost traditional fear of its dangers and occasional mishaps have prevented its use by some and dampened the ardor of others who at first used it enthusiastically.

Corning (1885) was the first in our knowledge to produce subarachnoid anæsthesia in dog and man. By injecting 3 per cent cocaine solution, he obtained anæsthesia of the lower extremities and thought the anæsthetic was carried into the cord by the circulation. Bier (1899) reported a series of 6 cases in which spinal anæsthesia (cocaine) was used in clinical surgery for the first time. He and an assistant also tried it on themselves and described their sensations. Tuffier and Hallion (1900) standardized the technique of clinical spinal anæsthesia. Doenitz (1903), Klapp (1904), and Heineke and Laewen (1906) are others among the earlier investigators of this subject.

The toxicity of cocaine solution retarded the general adoption of this mode of anæsthesia and, even after the advent of the less toxic agents such as stovaine and novocain, it was used only sporadically due to the fear of reactions which were imperfectly understood. From 1906 to 1928 occasional articles on the subject appeared in the literature, most of them clinical (Pauchet, 1920; Delmas, 1920; Soupalt, 1923; Babcock, 1925; Martin and Arbuthnot, 1926; Featherstone, 1924; Gray and Parsons, 1911; Ockerblad, 1927), a few experimental (Smith and Porter, 1915; Schilf and Ziegner, 1924; Bower, Wagoner, and Clark, 1926); but this method of anæsthesia was used routinely by only a few in this country and abroad.

In 1928, Pitkin came forward with his "controllable spinal anæsthesia" involving the use of solutions lighter or heavier than the spinal fluid. This was the stimulus for numerous articles which in turn led to the widespread use of spinal anæsthesia clinically. Among the articles appearing in 1928 were those of Koster, Evans, Labat, Leriche, Isenberger, and Rygh. Koster, using neocaine (novocain), advocated its use in surgery of the head, neck and thorax. Evans discussed the technique, and the treatment of complications. Labat, one of the pioneers in this field, also used neocaine and emphasized the importance of the Trendelenburg position to prevent anæmia of the brain. Isenberger and Rygh discussed the untoward reactions, and Leriche the technique of administration and control of reactions. These articles were followed in 1929 by those of Sise, Domenech, Koster, and Stout.

In the last few years, we have found attempts to place the subject of spinal anæsthesia upon a more rational basis by experimental investigation (Ochsner and Gage, 1930; Vehrs, 1931; Ferguson and North, 1932; Bower, Clark, Wagoner, and Burns, 1932; Cotui and Standard, 1932; Seevers and Waters, 1932). The present study was undertaken several years ago in an attempt to throw some light upon some controversial questions regarding this method of anæsthesia. We were particularly interested in the following points: the distribution of the anæsthesia in relation to the volume and concentration of the anæsthetic solution, and to the position of the patient; the behavior of the blood pressure with special reference to the cause, prevention, and treatment of its fall; the cause of death following spinal anæsthesia with special reference to the relation of blood pressure decrease to respiratory failure, and to the prevention of these; the

TABLE I.—EFFECT OF VOLUME ON SPREAD OF DYE IN CADAVERS, HORIZONTAL POSITION

No.	C.cm. injected	Site of injection	Pressure of spinal fluid (cm. water)	Upper level of stain
26	1	1-2 L	8	10 T
7	2	1-2 L	4	8 T
19	2	1-2 L	10	9 T
9	3	1-2 L	10	6 T
15	3	1-2 L	6	7 T
6	4	1-2 L	3	4 T
21	4	1-2 L	4	7 T
12	6	1-2 L		2 T
16	6	1-2 L		5 T
10	8	1-2 L	10	Brain stem

TABLE II.—EFFECT OF POSITION ON SPREAD OF DYE IN CADAVERS

No.	C.cm. injected	Site of injection	Pressure of spinal fluid (cm. water)	Upper level of stain	Position
2	2	2-3 L		3 L	Horizontal
22	2	2-3 L	8	9 T	Horizontal
3	2	2-3 L		12 T	Horizontal
23	2	2-3 L	4	8 T	Horizontal
24	2	2-3 L	8	6 T	Head lowered after injection
18	2	2-3 L	10	9 T	Head lowered after injection
13	2	2-3 L	10	7 T	Head lowered after injection
29	2	2-3 L	13	9 T	Head lowered 20° before injection
11	2	2-3 L	11	4 T	Head lowered 20° before injection
5	2	2-3 L	9	6 C	Head lowered 40° before injection
4	2	2-3 L	14	7 C	Head lowered 40° before injection
32	2	2-3 L	2	12 T	Head raised

effect of spinal anæsthesia upon intestinal tone and motility with special reference to the mechanisms involved and the clinical applications. The methods of study included injection of cadavers, clinical observations, and animal experimentation.

DISTRIBUTION OF ANÆSTHESIA

Cadaver experiments. Method: In order to simulate living conditions within the subarachnoid space as nearly as possible, it was first filled through a lumbar puncture needle with normal saline solution until the pressure as recorded on a water manometer registered somewhere between 13 and 130 millimeters of water. A dye (Loeffler's methylene blue) was then injected through the same needle and the brain and cord were later exposed to determine its distribution. The conditions of the experiments were varied as to the site of injection (first, second, or third lumbar interspaces); the volume injected (1, 2, 4, 6, or 8 cubic centimeters); whether the solution was reinjected or not; and the position on the table (horizontal, head raised, head lowered before injection, or head lowered after injection); only one factor being varied at a time.

In a series of 38 cadavers, many individual variations as to these points were found but the following generalizations can be made. The upward spread of the dye was directly proportional to the volume injected (Table I). Aspiration and reinjection were equivalent to injection of a larger volume without reinjection but were not as effective in spread as when the larger volume was injected at one time. The extension upward increased with depression and decreased with elevation of the head of the table, the amount being proportional to the degree of angulation (Table II). Varying the site of injection one or two spinal segments had only slight effect upon the spread, the difference usually equalling the distance between the points of injection, and sometimes being more than balanced by other factors (Table III). Variations in the amount of pressure existing within the subarachnoid space within the limits of normal living conditions (13 to 130 millimeters of water) seemed to have little or no effect on the extension of the dye, although it is conceivable that excessive pressures might retard and lowered pressures accelerate the diffusion (Tables I, II, and III). These generalizations represent tendencies only, for individual variations and exceptions were frequent making it impossible accurately and definitely to foretell the spread in any given case—rather one could but approximate the tendency in relative terms.

Clinical observations. In a series of 155 spinal anæsthesias given at the University Hospital, careful checks were made of the

weight of novocain (or neocaine) given, the volume of spinal fluid in which it was dissolved, the site of injection, the position of the patient, the height of anæsthesia (analgesia), the pulse, the blood pressure (before and several times during the course of the anæsthesia), the reactions, and finally the medication (if any) given (Table IV). For the moment we are concerned only with the height of anæsthesia in relation to volume, weight (concentration), position of the patient, and site of injection. Stout (1929) has published charts giving definite levels of anæsthesia for certain volumes and concentrations of solution. Our work has shown that, although certain generalizations can be made, any accurate prediction as to the level of anæsthesia under given conditions is impossible. As in the cadaver experiments, the most striking observation was the great variability in individual cases. For instance, in this series, injections of 4 cubic centimeters reached anywhere from the tenth thoracic segment to the brain stem, 3 cubic centimeters from the twelfth thoracic to the first cervical, 2 cubic centimeters from the tenth thoracic to the first cervical, and 1 cubic centimeter from the twelfth thoracic to the first cervical segments. Furthermore, the average for each of these volumes was about the same: fifth, fourth, fourth to fifth, and fourth to fifth thoracic segments, respectively. This would indicate that, within a limited variation, the level of anæsthesia was independent of volume in spite of the mechanical mixing. However, when one used enormous volumes (6 to 8 cubic centimeters or more) or if one reinjected several times, noticeable increase in anæsthesia level resulted (Table IV).

If the level of anæsthesia is now checked against the weight of novocain used, marked individual variations are again seen. For instance, 300 milligrams reached anywhere from the seventh thoracic segment to the brain stem, 200 milligrams from the tenth thoracic to the brain stem, and 150 milligrams from the twelfth thoracic to the eighth cervical segments. However, here the averages were much more distinctive, that for 300 milligrams being seventh to eighth cervical, for 200 milligrams fourth thoracic, and for 150 milligrams fifth to sixth thoracic segments. Again, the average weight of novocain for each of the common volumes used (4, 3, 2, and 1 cubic centimeters) was about the same (190, 175, 192, and 190 milligrams, respectively). This would indicate that the level of anæsthesia was proportional to the concentration of solution as long as the volume was within the range of 1 to 4 cubic centimeters. To put it another way, although the level of anæsthesia in any given case can not be accurately foretold for any given dosage (volume and weight), one can expect higher levels (cervical or above) by means of greater mechanical mixing (volumes over 4 cubic centimeters or reinjection of smaller volumes) or by higher concentration of solutions (300 milligrams or more) or by both; whereas dosages of 150 to 200 milligrams dissolved in 1 to 4 cubic centimeters of spinal fluid may be expected to reach to the mid-thoracic region, although individual cases may go considerably higher or lower. This tendency of moderate dosages to remain below the mid dorsal region, we believe to be a definite safety factor as we shall attempt to bring out later.

TABLE III.—EFFECT OF SITE OF INJECTION ON SPREAD OF DYE IN CADAVERS

No.	C.cm. injected	Site of injection	Pressure of spinal fluid (cm. water)	Upper level of stain	Position
37	2	1-2 L	6	6 T	Head lowered
30	2	2-3 L	10	9 T	Head lowered
35	2	3-4 L	8	8 T	Head lowered
33	2	1-2 L	6	8 T	Horizontal
34	2	2-3 L	8	9 T	Horizontal
27	2	3-4 L		1 L	Horizontal
38	2	3-4 L		12 T	Horizontal

Effect of position on the level of anæsthesia. In most of our clinical cases the head was lowered after injection, in some before injection, and in a few it was raised after injection. Taking into consideration the wide variations in levels independent of position, there is still evidence to corroborate the conclusions arrived at in our cadaver experiments: that the upper level of anæsthesia is increased by lowering (more so before injection) and decreased by raising the head of the table (Table IV). If one desires to limit the spread of anæsthesia after injection,

it is best to maintain the horizontal position for 5 or 10 minutes, after which the anæsthetic solution is presumably fixed by the nervous tissue and little or no additional spread takes place. The head may then be lowered to overcome the effects of any fall in blood pressure.

Relation of the site of injection to the level of anæsthesia. Most of our clinical cases were injected in the first, second, or third lumbar interspaces. Within these limits no definite effect upon anæsthesia level was noticeable or if there were any it was so slight as to be overshadowed by the greater individual variations noted in these cases. However, greater variations in the position of the injection (such as mid dorsal) would no doubt show definite differences in anæsthesia level, the amount depending upon the height of injection. These findings are again in accord with those noted in our cadaver experiments.

Distribution of anæsthetic solution in laboratory animal experiments. In order to determine the course taken by an anæsthetic solution injected subarachnoidally, a series of experiments was made, cats, rats, dogs, and rabbits being used. In all, 18 cats, 15 rats, 2 dogs, and 19 rabbits were used. At first an attempt was made to inject through the unbroken skin but, due to the uncertainty of this method, it was replaced by laminectomy and subsequent injection under direct vision. Two per cent novocain solution, colored with methylene blue so that the distribution could be determined postmortem, was injected in various volumes. Following are a few of the typical protocols.

Cat 11. Ether anæsthesia was administered only sufficient to allow laminectomy in the upper lumbar region. One half cubic centimeter novocain methylene blue solution was injected subarachnoidally at 9:45 a.m. Analgesia was effected to upper cervical region at 9:50 a.m. Another half cubic centimeter was injected at 9:55 a.m. Animal died one half minute later, respiration ceasing first and then the heart gradually failing. At postmortem examination a blue stain was noted around the entire cord and spinal nerve roots, on all of ventral surface of medulla but only on the inferior half of the dorsal surface (in cisterna magna), on the entire ventral surface of the brain stem covering all the cranial nerves as far as the olfactory nerve. Section of cord, medulla, brain stem, and spinal nerve roots at various levels showed no penetration of color into the substance of the nervous tissue.

Rat 1. Ether anæsthesia was administered to allow laminectomy in the upper lumbar region. Injection of 1 cubic centimeter of novocain methylene blue solution was done subarachnoidally. Sudden respiratory death occurred during injection. At postmortem examination a blue color was noted around the entire cord, all spinal nerve roots, the entire brain, and all the cranial nerves. Sections failed to show color within nervous substance.

These experiments showed the usual course of the solution as extending upward around the cord and nerve roots, then around the medulla following first along the ventral surface of the medulla and brain stem, surrounding the cranial nerve roots, and to a lesser extent along the dorsum of the medulla. With increasing volume, the dorsum of the upper medulla, cerebellum, and cerebral cortex were finally reached in the order named. If the extension of the color were an indication of the extension of the novocain itself, the failure of the blue to penetrate the nervous substance was not in accord with the production of anæsthesia and with the death of the animal. That the novocain penetrated further into the nervous tissues than the dye with which it was in solution was the inevitable conclusion. To obtain direct evidence of this as well as to determine the exact distribution within the nervous tissues, a method of recognizing novocain within the substance of the latter was sought. This was accomplished with the aid of Dr. Meyer Beber, of the department of Biochemistry at the University of Nebraska, College of Medicine. A brief description of the method follows:

Diazo color reaction for novocain within the tissues. Two per cent novocain solution (uncolored) is injected subarachnoidally in the experimental work. At autopsy the brain and cord with as much of the cranial and spinal nerve roots as possible are removed and placed into a beaker of cold 5 per cent sodium nitrite solution. After a few minutes hydrochloric acid (one-tenth dilute) is added in the proportion of one of the latter to five of the former solution. This liberates nitrous acid which in turn diazotizes the novocain. It is essential that the solution be kept cold for this reaction. After 5 minutes, the specimens are washed in distilled water and transferred to a 5 per cent alcoholic solution of beta naphthol. The tissue

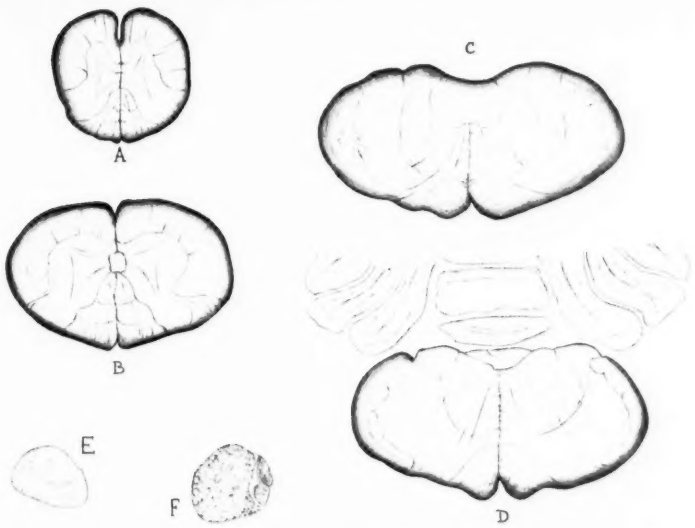

Fig. 1. Drawings of transverse sections of cord, medulla, and nerve roots of rat, showing penetration of novocain solution into nervous tissue as indicated by the Diazo color reaction. *A*, Thoracic cord (×8); *B*, cervical cord (×8); *C*, open portion of medulla (×8); *D*, open portion of medulla, a little higher than C (×8); *E*, control nerve root (×32); *F*, nerve root showing Diazo reaction (×32).

containing novocain takes on an orange red color which is greatly intensified by transferring to a weak solution (2 per cent) of sodium hydroxide. The color fades if placed in water or carried through the usual solutions used in preparing paraffin sections. For section work it was found best after bringing out the color reaction to fix the tissue in 10 to 20 per cent formalin for 15 to 20 minutes, freeze, and cut thick (50 μ). Control experiments, in which either distilled water was injected into the subarachnoid space or no injection made at all, failed to produce a similar color reaction when subjected to the same process. The following protocols are typical of those experiments in which this method was used.

Rat 3. Ether anæsthesia was given only sufficient to allow laminectomy in the upper lumbar region. One half cubic centimeter of 2 per cent novocain solution was injected into the subarachnoid space. Sudden cessation of respiration was followed shortly by death during last part of injection. The brain, cord, and nerve roots were removed and tested for novocain by the method described above. Grossly the typical orange red color covered the entire cord, all the spinal nerve roots, the entire brain including even the dorsum of the medulla, cerebellum, and cerebral cortex, and all the cranial nerves. Frozen sections showed the color penetrating the substance of the cord and the brain for a variable distance (approximately ¼ millimeter), and extending completely through the nerve roots (Fig. 1, a, b, c, d, e, f).

The open portion of the medulla (where the respiratory and vasomotor venters are supposedly located) was stained well into the substance but the uppermost portion, under cover of the cerebellum, was not stained on the dorsum, there apparently being a sharp dividing line produced by a membrane (pia) separating the fourth ventricle from the subarachnoid space. Possibly the larger amount of spinal fluid in the fourth ventricle so diluted the penetrating novocain as to make its color reaction invisible.

Rat 11. The same procedure was used. One cubic centimeter of 2 per cent novocain solution was injected into the subarachnoid space. The animal died suddenly after ⅓ cubic centimeter was injected, the respiration ceasing first. The brain, cord, and nerve roots were treated as stated to bring out the color reaction. Grossly the orange red color was heavy around the entire cord, spinal nerve roots, ventral surface of the medulla and brain stem, and all the cranial nerves. It was lighter over the dorsum of the medulla and very faint over the dorsum of the cerebellum and cerebral cortex. Sections showed a penetration into the nervous tissue similar to that in rat 3.

TABLE IV.—CLINICAL CASES

Patient and operation	Mgm. novocain	Vol. c.cm.	Position of patient	Anæsthesia level	Pulse	Blood pressure
1. Salpingectomy	150	4	l.b.	2 T		130/78-110/70-88/65-120/70
2. Appendectomy	150	4	l.a.	3 T	84-78-70	128/78-100/56-90/48-100/50
3. Appendectomy	150	4	l.a.	2 T	142-102-96	122/70-100/60-98/64
4. Appendectomy	150	4-R	l.a.	4 T		90/70
5. Appendectomy	150	4	l.a.	10 T		
6. Appendectomy	150	3	l.a.	2 T	112-148	112/42-88/58
7. Perineorrhaphy	150	4	l.a.	3 T	112-72-96	120/76-70/50-120/84
8. Hammer toe	150	1.25	l.a.	10 T	80-88	128/86-140/82-126/76
9. Appendectomy	150	2.25	l.a.	7 T	80-100-84	110/80-128/84-122/76
10. Appendectomy	150	2	l.a.	2 T	82-108-88	134/70-100/54-120/70
11. Patellar suture	150	1.5	l.a.	2 T	80-78-96-64	110/60-86/52-104/66
12. Hæmorrhoidectomy	150	4	l.a.	5 T	80-78-80	120/90-110/70-90/70
13. Cholecystectomy	150	4	l.a.	10 T	110-160	118/75-117/75
14. Hæmorrhoidectomy	150	4	l.a.	3 T	80-90-126	96/60-110/70-75/54
15. Herniorrhaphy	150	2.5	l.a.	2 T	92-78-74	130/78-98/66-96/60
16. Appendectomy	150	3	l.a.	6 T	78-96-84	88/70-102/66
17. Hysterectomy	150	1.5	l.a.	7 T	80-70-90-60	106/72-130/74-106/60
18. Gastro-enterostomy	150	2	l.a.	7 T	56-76-66-72	82/48-112/64-98/52
19. Cystotomy	150	1	l.a.	7 T	90-90-100	180/90-160/90-160/90
20. Suspension	150	3.5	l.a.	4 T	86-76-74	84/62-80/60-82/60
21. Herniorrhaphy	150	3	l.a.	4 T	70-75-75	120/75-100/60-105/70
22. Wound suture	150	3	l.a.	7 T	100-120-140	135/85-105/80-150/90
23. Appendectomy	150	3	l.a.	4 T	65-80	115/80-130/90
24. Herniorrhaphy	150	3	l.a.	5 T	80-65	130/80-120/70-110/70-106/60
25. Appendectomy	150	4	l.a.	4 T	80-100	128/80-138/80-90/60-100/60
26. Herniorrhaphy	150	4	l.a.	7 T	80-100-80	130/70-95/60-70/50-70/40-80/50-85/55
27. Herniorrhaphy	150	4	l.a.	7 T	80-60-75	130/80-60/40-70/40-80/50
28. Appendectomy	150	3	l.a.	7 T	100-120-110	115/70-135/70-115/70
29. Open reduction	150	3	l.a.	12 T	70-70-70	120/70-130/70-130/70
30. Appendectomy	150	.5	l.a.	12 T	125-128-100	125/70-115/70-98/70-85/55
31. Urethrotomy	150	6	l.a.	10 T	70-75-70	125/70-130/80-130/70
32. Appendectomy	150	1	l.a	2 T	90-100-78	120/80-100/70-75/40
33. Bunion	150	3	l.a.	3 T	80-70-65	130/80-120/60-120/60
34. Cystotomy	150	3	l.a.	1 T	116-100-80	125/70-90/60-100/60
35. Appendectomy	150	3	l.a.	5 T	66-66	115/70-125/80-115/70
36. Drainage	150	2.5	l.a.	9 T	116-112	120/78-112/72
37. Appendectomy	150	1.5	l.a.	5 T	76-60-64	92/50-100/55-110/72
38. Appendectomy	150	1.2	l.b.	9 T	106-98	120/84-130/70
39. Appendectomy	150	3	l.a.	1 C	90-100-90	130/80-115/60
40.	150	3.5	l.a.	8 T	72-84	80/38-102/76
41.	150	3	l.a.	2 T	88-120	126/64-112/64
42.	150	1	l.b.	7 T	88-68	110/72-98/60
143. Cystotomy	150	4	l.a.	7 T	104-100	138/84-118/74
44. Cystotomy	150	3.5	l.a.	6 T	120-104	152/100-124/70

TABLE IV.—CLINICAL CASES—Continued

Patient and operation	Mgm. novocain	Vol. c.cm.	Position of patient	Anæsthesia level	Pulse	Blood pressure
45. Appendectomy	150	1	l.a.	4 T	132-96	124/70-90/56
46. Appendectomy	150	4	l.a.	5 T	112-60-76	90/60-100/60
47. Hæmorrhoidectomy	150	4	l.a.	10 T	9-90	100/62-112/78
48. Appendectomy	150	1	l.a.	2 T	114-100-128	118/65-110/60-128/65
49. Appendectomy	150	1	l.a.	1 T	148-160-108	117/58-128/62-130/60
50. Hysterectomy	150	1	l.a.	4 T	112-106-114	130/80-110/72-94/60-95/58
51. Appendectomy	150	1	l.a.	8 T	80-112-96	100/64-94/60-120/82
52. Hysterectomy	150	.9	l.a.	1 T	100-84	108/78-95/80-94/70
53. Hysterectomy	150	.5	l.a.	7 T	128-116	110/70-110/70
54. Appendectomy	150	1	l.a.	9 T	72-64-64	140/80-140/80
55. Drainage	150	1	l.a.	0		140/80-130/75
56. Drainage	150	1	l.a.	11 T	80-76	140/90-130/85
57. Appendectomy	150	1.1	l.a.	6 T	64-60	118/78-152/84
58. Appendectomy	150	.8	l.b.	9 T	116-112	125/65-105/70-120/70
59. Suspension	150	1	l.a.	4 T	72-108-92	140/80-108/64-110/68
60. Appendectomy	150	2	l.a.	2 T	84-104-72	110/65-106/62
61. Appendectomy	150	2-R	l.a.	2 T	160-100-92	110/74-90/62-70/60
62. Hysterectomy	150	.7	l.a.	11 T	128-140-152	152/90-120/40
63. Perineorrhaphy	150	1	l.b.	2 T	160-112	114/82-98/65-94/68
64. Herniorrhaphy	200	4	l.a.	3 T	120-70	98/60-106/64
65. Salpingectomy	200	2.5	l.a.	3 T	108-136-72	136/80-78/40-106/60
66. Herniorrhaphy	200	2.5	l.a.	3 T	136-132-108	152/86-114/82-140/82
67. Cholecystectomy	200	3	l.a.	3 T	60-50-60	96/64-78/46-80/50
68. Appendectomy	200	2	l.a.	4 T	84-72-72	130/78-138/80-136/78
69. Amputation	200	2	l.a.	10 T	80-93-85	120/75-113/78-115/75
70. Perineorrhaphy	200	2	l.a.	4 T	80-75-85	115/85-120/80
71. Appendectomy	200	4	l.a.	3 T	60-72-66	128/52-110/60-122/78
72. Appendectomy	200	4	l.a.	3 T	60-66-66	128/52-110/60-118/76-122/78
73. Anal repair	200	4	l.a.	5 T	100-138-72	140/72-110/76-94/60-102/68
74. Perineorrhaphy	200	4	l.a.	1 T	100-108-102	116/76-124/94-108/82-112/86
75. Appendectomy	200	1	l.a.	7 T	74-80-76	122.78-138/64-118/68
76. Perineorrhaphy	200	2	l.a.	4 T	98-85-96	140/90-145/95-142/100
77. Cholecystectomy	200	2	l.a.	7 T	84-140-128-92	130/90-112/76-126/76
78. Suspension	200	2	l.a.	4 T	84-120-80	102/74-100/66-110/70
79. Hysterectomy	200	2.5	l.a.	7 T	92-64-68-60	92/60-86/64-84/64
80. Hysterectomy	200	2	l.a.	3 T	132-96-100	154/80-122/72-128/58
81.	200	4	l.a.	7 T	76-100-100	130/85-150/90-140/82
82. Appendectomy	200	4	l.a.	4 T	70-70	120/70-154/90-130/90
83. Appendectomy	200	4	l.a.	7 T	72-85-85	124/84-100/60-130/80
84. Salpingectomy	200	4	l.a.	7 T	86-100-85	120/56-114/60-120/60
85. Appendectomy	200	4	l.a.	8 T	124-108-104	110/80-108/78-100/80
86. Herniorrhaphy	200	4	l.a.	10 T	60-65-56	90/60-106/60-96/60
87. Appendectomy	200	3	l.a.	4 T	88-72-68	130/69-146/88-136/74
88. Cholecystectomy	200	4	l.a.	4 T	95-50-60-100	160/90-180/60-200/70-130/70

TABLE IV.—CLINICAL CASES—Continued

Patient and operation	Mgm. novocain	Vol. c.cm.	Position of patient	Anæsthesia level	Pulse	Blood pressure
89. Herniorrhaphy	200	4	l.a.	4 T	72-100-120	140/80-70/60-120/60
90. Appendectomy	200	3	l.a.	4 T	65-120-100	110/70-85/60-90/60
91. Appendectomy	200	3	l.a.	4 T	75-90-90	110/70-85/60-115/70
92. Appendectomy	200	3	l.a.	4 T	80-100	130/90-145/100
93. Salpingectomy	200	3	l.a.	2 T	70-90-65	95/60-80/50-125/70
94. Herniorrhaphy	200	4	l.a.	8 T	60-100-80	180/80-120/80-130/80-210/90
95. Appendectomy	200	4	l.a.	4 T	80-100-80	115/75-130/80-130/90
96. Salpingectomy	200	4	l.a.	10 T	90-100-110-90	130/75-80/60-100/60-150/80
97. Skin graft	200	4	l.a.	2 T	80-70-60	140/90-120/90-120/80
98. Salpingectomy	200	3	l.a.	1 C	70-90-70	105/70-140/90-115/80
99. Colostomy	200	4	l.a.	3 T	60-60-60	100/60-95/60-100/60
100. Exploratory	200	4	l.a.	2 T	80-84-85	140/80-80/50-95/70
101. Suspension	200	1	l.a.	3 T	60-84-72	90/60-100/70-94/64
102. Cholecystectomy	200	1	l.a.	1 T	96-60	130/80-76/—
103. Hysterectomy	200	1	l.a.	5 T	140-76-106	162/115-85/75-98/75
104. Cholecystectomy	200	3.2	l.a.	2 T	96-104-80	114/76-148/94-102/70
105. Appendectomy	200	2	l.a.	1 T	86-79	150/85-110/75-100/75
106. Hysterectomy	200	4	l.a.	1 C	120-88	144/90-126/87-110/70-118/76
107. Prostatectomy	200	4	l.a.	5 T	104-76-76	124/84-74/56-60/—-82/68
108. Herniorrhaphy	200	4.5	l.a.	4 T	100-76-132	124/70-90/60-94/62-126/80
109. Perineorrhaphy	200	1	l.a.	4 T	85-100	110/65-102/75
110. Salpingectomy	200	1	l.a.	1 T	100-95-88	100/55-70/40-85/55-92/50
111. Hysterectomy	200	1	l.a.	4 T	88-120-100	140/80-80/70-90/70-130/80
112. Appendectomy	200	2.5	l.a.	6 T	90-100-90	140/105-110/85-118/84
113. Suspension	200	1	l.a.	10 T	84-96-88	97/58-105/60-98/62-100/66
114. Hysterectomy	200	1.8	l.a.	4 T	72-108-92	90/60-72/58-130/90
115. Herniorrhaphy	200	1	l.a.	2 T	68-88-72	135/90-112/76-158/95-118/82
116. Cholecystectomy	200	2.5	r.a.	7 T	76-116-68	100/80-72/50-78/60
117. Cholecystectomy	200	.5	r.a.	10 T	116-92-120	100/70-92/70-90/55
118. Perineorrhaphy	200	1.5	l.a.	4 T	120-116-80	138/88-118/68
119. Salpingectomy	200	1.4	l.a.	8 T	116-120-104	100/70-80/55-106/80
120. Salpingectomy	200	1	l.a.	5 T	104-92	112/78-105/70-118/78
121. Drainage	200	1.4	l.a.	10 T	108-120-92	100/60-60/46-48/42
122. Curettage	200	1.1	l.a.	2 T	88-84	80/50-85/52
123. Salpingectomy	200	1	l.a.	10 T	100-92-88	120/84-136/94-120/86
124. Appendectomy	200	1	l.a.	5 T	88-136-124	115/76-135/80-132/74
125. Appendectomy	200	2.6	l.a.	7 T	100-108	100/68-90/64
126. Cholecystectomy	300	4	l.a.	1 T	96-78	160/100-84/62
127. Herniorrhaphy	300	4	l.a.	Brain stem	84-156-90	102/64-138/106-92/72-110/90
128. Herniorrhaphy	300	4	l.a.	6 T	80-78-80	142/90-90/70-115/70
129. Rib resection	350	8	l.a.	Brain stem	80-64-66	78/60-72/60-60/42-74/54
130. Perforated ulcer	300	3	l.a.	4 T	115-140-150	130/75-95/65-100/65
131. Interposition	300	1	l.a.	1 C	80-70	160/90-80/50-75/45-40/—
132. Salpingectomy	300	1	l.a.	2 T	80-90-100	110/70-104/80-150/110-135/100

TABLE IV.—CLINICAL CASES—Continued

Patient and operation	Mgm. novocain	Vol. c.cm.	Position of patient	Anæsthesia level	Pulse	Blood pressure
133. Hysterectomy	300	1.5	l.a.	1 C	80-90-80	130/90-100/80-85/60-90/50
134. Appendectomy	300	1	l.a.	1 C	90-60-70	140/90-90/50-105/70
135. Hysterectomy	300	1	l.a.	8 C	70-80-85	110/80-70/50-60-80/50-150/80
136. Hysterectomy	300	4	l.a.	4 T	108-62-92	140/90-86/58-70/40-86/60
137. Salpingectomy	300	2	l.a.	4 T	110-120-130	140/80-96/60-120/80
138. Hysterectomy	300	2	l.a.	1 C	68-104-92	130/85-102/80-112/76-106/72
139. Hysterectomy	300	2	l.b.	3 T	122-80-112	126/85-104/75-130/85
140. Hysterectomy	300	1	l.a.	7 T	124-146-116	132/80-142/82-126/84
141. Jaw abscess	400	8	l.a.	Brain stem	70-70	122/85-105/75-80/50
142. Ovarian cyst	400	4	l.a.	7 T	94-80-105	125/96-100/79-85/62-92/70
143. Cholecystectomy	225	6	l.a.	1 C	88-98	130/78-85/60
144. Exploration	225	6	l.a.	Brain stem	64-68	110/60-86/50-86/52
145. Cæsarean section	225	4	l.a.	2 T	116-96-120	124/64-90/42-130/58
146. Perineorrhaphy	126	6	l.a.	7 T		
147. Hysterectomy	120	4	l.a.	10 T		
148. Salpingectomy	225	4 R	l.a.	8 T		
149. Appendectomy	225	6 R	l.a.	2 T	144-90	122/66-115/62
150. Appendectomy	225	4	l.a.	1 T	62-90-105	126/80-140/80-134/70
151. Herniorrhaphy	225	4	l.a.	4 T	68-72-72	168/100-130/98-140/90
152. Suspension	225	4	l.a.	2 T	60-106-80	106/62-98/58-110/78
153. Hysterectomy	225	4	l.a.	3 T	84-106-102	102/68-128/90-102/76-90/60
154. Hysterectomy	225	4	l.a.	1 T	86-72-60	134/78-98/56-184/98
155. Osteotomy	100	2	l.a.	2 T	135-144	110/66-102/66

l.b.—Head lowered before injection. l.a.—Head lowered after injection. r.a.—Head raised after injection.

These experiments show the same general distribution as seen in those in which methylene blue was used as the coloring agent, but in addition indicate that the anæsthesia may be the result either of action upon the nerve roots or upon the sensory tracts within the cord and brain. They also show the possibility of the anæsthetic solution reaching the vital medullary centers if sufficient volume is used.

THE BLOOD PRESSURE IN SPINAL ANÆSTHESIA

The most disconcerting complication of spinal anæsthesia has been the marked fall in blood pressure which so often occurs. The real cause of this fall has been the subject of considerable controversy. Doenitz (1903) and Klapp (1904) thought it due to the absorption of the drug into the circulation. Tuffier and Hallion (1900) thought it due to action on the cord or nerve roots since they noted rise in blood pressure from stimulation of the peripheral nerves (splanchnics) after the administration of spinal anæsthesia.

Heineke and Laewen (1906) found with subarachnoid injections of cocaine an immediate, intensive, long lasting fall in blood pressure and frequently immediate death; with intravenous injections an immediate but short fall in blood pressure and only with the largest doses death. Intramuscular injections produced no noticeable changes in blood pressure. They placed a ligature about the upper thoracic cord and found only slight fall in blood pressure following injection below the ligature but intense fall (30 to 50 per cent) and death following injection above the ligature. They concluded that the drop in blood pressure was due to direct action upon nerve centers rather than to absorption of the drug into the circulation. Smith and Porter (1915) thought the drop due to paralysis of the splanchnics, not to paralysis of the bulbar

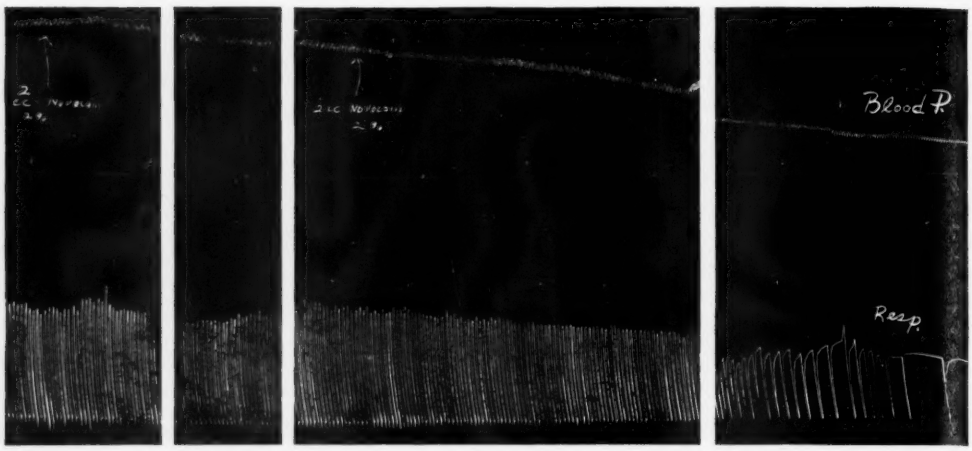

Fig. 2. Tracings showing independence of the fall in blood pressure and changes in costal respirations following subarachnoid injection of novocain solution. Dog.

vasomotor centers, since they found the greatest percentage fall when the anæsthetic was confined to the thoracic as compared to the cervical and lumbar regions.

Schilf and Ziegner (1924) injected subarachnoidally the various regions of the cord isolated by ligatures and found only slight fall in blood pressure with injections of novocain confined to the lumbar region but a much greater fall (still compatible with life) with injections confined to the thoracic region. Injections above the upper thoracic region caused the animal to stop breathing, the blood pressure remaining up for a minute or so and death ensuing unless artificial respiration was instituted. They concluded that the fall was due to action upon the preganglionic fibers of the thoracic region (splanchnics). Ferguson and North (1932) found only slight drop in pressure by sectioning the splanchnics and the usual profound fall by subarachnoid anæsthesia following section. By injection of novocain solution subarachnoidally below a ligature placed at the level of the fifth thoracic segment they found only a moderate decrease in blood pressure (19 per cent); whereas injection above the ligature produced a most intense fall (37 per cent additional or 56 per cent in all). Kremer and Wright (1932) found similar results from sectioning the splanchnics. Bower, Clark, Wagoner, and Burns (1932) also found the greatest fall in blood pressure with injections into the upper thoracic and cervical regions, but explained its occurrence by the interference with respiration and the loss of the sucking action of the chest upon the right side of the heart—the marked fall in blood pressure being then due, according to them, to myocardial failure instead of vasomotor paralysis. A somewhat similar view was expressed by Seevers and Waters (1932). Cotui and Standard (1932) found direct paralysis of respiratory and vasomotor centers by cisternal injections of small quantities of novocain solution.

Out of this maze of evidence one fact stands forth; the effect on the blood pressure is due to local action upon the nerve roots, cord, or medulla and not to absorption into the circulation, since much larger doses than are used in spinal anæsthesia are necessary to produce even slight fall in blood pressure when injected intravenously. Whether this effect is due to paralysis of the vasomotor center in the medulla, or vasomotor fibers within the cord or nerve roots, or whether it is secondary to myocardial weakness resulting from interference with respiratory movements by the action of the anæsthetic on intercostal or phrenic nerves, are questions in which we were particularly interested and have attempted to answer by animal experimentation. A series of

2 dogs and 17 rabbits were used for this purpose. We first wished to test out the theory of the fall in blood pressure being due to cardiac weakness resulting from interference with respiration. The following are a few typical protocols.

Dog 1. Ether anæsthesia, just sufficient to permit the necessary surgical procedures. The right carotid artery was cannulated and connected with a mercury manometer, the pointer of which was made to record upon a kymographic drum. Costal respirations were also recorded by means of a pneumograph and air tambour. A laminectomy was done in the upper lumbar region, thus exposing the dura. With a fine needle and Luer syringe, 2 cubic centimeters of 2 per cent novocain solution colored with methylene blue were injected into the subarachnoid space, the needle being pointed cephalad. There was considerable leakage around the needle so that only a part of the solution entered the subarachnoid space. After a short latent period there was a gradual fall in blood pressure, the respirations remaining unchanged. Five minutes later an additional 2 cubic centimeters were injected (again with leakage). This was followed by an additional but still gradual fall in blood pressure, the respirations still remaining about the same. Finally when the blood pressure reached 40 millimeters of mercury, the respiratory movements showed a slowing and decrease in amplitude, and at 20 millimeters stopped completely. Since the fall in blood pressure preceded by a considerable period any change in the respirations, it is difficult to see how the latter could be responsible for the fall noted in this case (Fig. 2).

Rabbit 4. Urethane and morphine anæsthesia. The trachea was cannulated and artificial respiration carried on throughout the experiment. Laminectomy was done in the lower thoracic and upper lumbar regions and 1 cubic centimeter of 2 per cent novocain colored with methylene blue was injected cephalad into the subarachnoid space. There was slight leakage about the needle. The typical fall in blood pressure took place (Fig. 3) in spite of the fact that artificial respiration was carried on, preventing any possible myocardial weakness due to interference with respiration. In fact, Bower, Clark, Wagoner, and Burns (1932) in their own experiments with myocardiographic tracings used artificial respiration and got the usual fall in blood pressure with spinal anæsthesia, making it difficult to see how the fall could be due to respiratory difficulty. In addition, the fall in pressure in their experiments occurred before changes in myocardiographic tracings, indicating independence of etiology.

The results in these experiments make untenable any theory placing the mechanism of blood pressure fall upon a basis of myocardial weakness, resulting from interference with

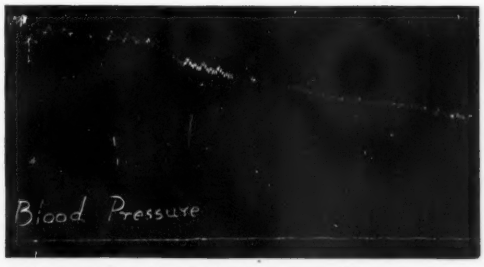

Fig. 3. The fall in blood pressure following a subarachnoid injection of 1 cubic centimeter of 2 per cent novocain solution in a rabbit under artificial respiration.

respiration. That the latter occurs in spinal anæsthesia, we agree (see below), but only in a concomitant relation to blood pressure fall rather than in one of cause and effect. We also grant that, with complete cessation of respiration, there is an additional sharp drop in blood pressure (after a preliminary anoxæmic rise) due to cardiac failure, but this is a terminal phenomenon and not the ordinary fall compatible with safe anæsthesia. Since in the experiments enumerated, there were also no appreciable changes in total blood volume, the usual fall in blood pressure must have been due to reduction in peripheral resistance, which means that the anæsthetic must have affected the vasomotor system. The only parts of the latter system accessible to the anæsthetic solution are the vasomotor center in the medulla or the vasomotor fibers within the cord or nerve roots. That it is possible for a solution to reach the interior of the cord and medulla as well as of the nerve roots has been demonstrated by our diazo test for novocain. In either event, the effect must be upon the medullary center or upon the preganglionic sympathetic fibers. This means that it is still possible after spinal anæsthesia to produce vasoconstriction and rise in blood pressure by stimulation of the postganglionic fibers or myoneural junctions, as with the use of ephedrine or adrenaline. Figure 4 is a record of an experiment demonstrating this latter point experimentally in animals. In our clinical cases are numerous examples of rise in blood pressure when adrenaline was injected following marked fall during the course of spinal anæsthesia (Cases 1, 7, 10, 11, 22, etc., Table IV).

Fig. 4. The effect of an intracardiac injection of 0.2 cubic centimeter of 1:1000 solution of adrenaline chloride upon the blood pressure which had fallen to 24 millimeters during spinal anæsthesia. Rabbit.

Boshamer (1925) has sectioned the white rami communicantes in the frog and noted dilatation of the peripheral vessels innervated. This is a most difficult procedure in the usual laboratory animals—the difficulties of exposure and recognition making it unfeasible. Instead, we have sought the accessible preganglionic fibers in our rabbit experiments. The animals were anæsthetized with urethane, which according to Heineke and Laewen (1906) does not affect the respirations, and morphine, and the carotid artery cannulated for blood pressure recording. Artificial respiration was used throughout in some animals, in others only when the respirations became depressed. The sympathetic chains in the neck and the splanchnics in the abdomen, both made up largely of preganglionic fibers, were exposed. We tried, but abandoned as unsatisfactory, the posterior approach for exposure of the splanchnics as described by Ferguson and North (1932). It is almost impossible by such a method to be certain of exposing the proper structures and the procedure possesses no advantage over high abdominal approach. In addition, the upper thoracic sympathetic chains were exposed by resecting a part of the chest wall on each side. These chains, of course, contained both preganglionic and postganglionic fibers. The effect upon the blood pressure of stimulating and sectioning these was noted as was also the effect of spinal anæsthesia, induced after sectioning.

The results (Fig. 5) were quite definite, though sometimes slight or moderate in degree. Stimulation of either preganglionic or postganglionic fibers caused a constriction of the peripheral vessels (ear vessels in case of cervical sympathetics) and a rise in blood pressure, and section of these nerves produced a corresponding dilatation and fall, a latent period of variable length first ensuing. After the cervical, splanchnic, and upper thoracic chains were sectioned and a spinal anæsthetic then given, there occurred an additional, more marked, but gradual fall in blood pressure, indicating that additional vasoconstrictor fibers were reached by the anæsthetic that were not caught in the sections. Whether the action of the novocain be upon the preganglionic vasoconstrictor fibers within the nerve roots or those descending within the cord is immaterial. Since the vasoconstrictor fibers emerge from the first thoracic to the third or fourth lumbar segments of the cord (Starling, 1930), it is obvious that the higher the anæsthetic reaches in the thoracic region the greater the number of vasoconstrictor fibers thrown out and the greater the resulting fall in blood pressure. The fall in blood pressure was gradual as long as the solution was kept below the medulla. When, however, but a few drops were injected into the cisterna magna so as to reach the medullary centers, there was a sudden, abrupt fall in blood pressure as well as a sudden cessation of respiration (Fig. 6), due to direct action upon the vital centers.

Until recently it has been held that the greatest dilatation occurred in the splanchnic region. However, Ferguson and North (1932) and Bower, Clark, Wagoner, and Burns (1932) have shown in dogs, by confining the anæsthetic solution to certain parts of the cord through the use of ligatures, that only a moderate fall in blood pressure took place when the anæsthetic was confined below the fifth thoracic segment (the splanchnics coming off from the fifth to twelfth thoracic), while a much greater fall occurred when the injection was made into the compartment above the fifth thoracic region. This was in harmony with the usual failure to note marked vasodilatation in the splanchnic area during the course of abdominal operations under spinal anæsthesia. They concluded that about one-third of the blood pressure change was due to dilatation in the splanchnic area, abdominal wall, and lower extremities, and two-thirds to vasodilatation in the head, neck, upper ex-

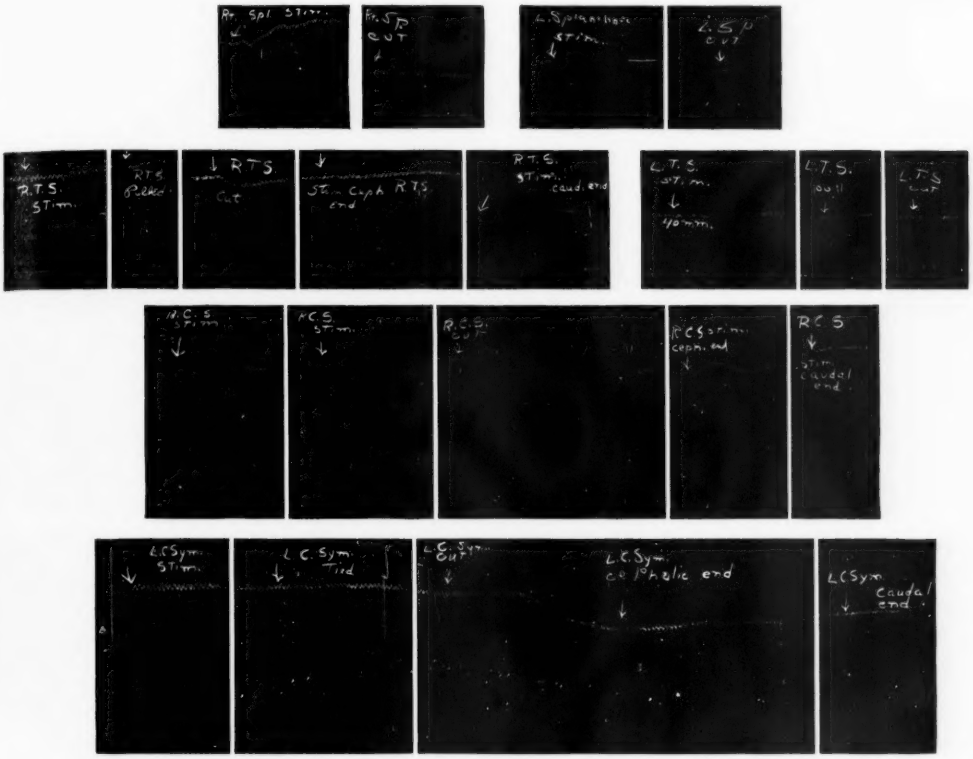

Fig. 5. The effects upon blood pressure of stimulating and cutting the right splanchnic, left splanchnic, right thoracic sympathetic, left thoracic sympathetic, right cervical sympathetic and left cervical sympathetic chains, in the order shown. The experiments were carried out on a rabbit.

tremities, thorax, and its contents. By checking postmortem the upper level of the stain, mixed with the novocain used in subarachnoid injections, with the blood pressure records during life, we have arrived at approximately the same conclusions from our animal experiments.

Interesting clinical data along the same lines may be obtained from Table IV by checking the blood pressure fall with the height of anæsthesia as determined by the pin test. In 55 cases in which the anæsthetic was confined below the fourth thoracic segment, the average fall in systolic pressure was nine points and in pulse pressure six points. The average fall in 70 cases, in which the upper level of anæsthesia was between the fourth thoracic and eighth cervical segments, was fifteen in systolic pressure and eighteen in pulse pressure. In 18 cases in which the anæsthesia reached higher than the eighth cervical segment, the average fall in systolic pressure was twenty-seven points and in pulse pressure fourteen points. It must be remembered that as the anæsthetic solution reaches higher in the cord a cumulative action results, the nerve roots being anæsthetized progressively from below upward and the descending fibers within the cord being anæsthetized so that segments of the cord below are affected. Perhaps the latter accounts for the relatively greater fall in the cervical region even though all vasoconstrictor fibers are given off from the cord below that region. These percentages do not give an exact picture of the amount of blood pressure drop because most of these cases had a preliminary injection of ephedrine which

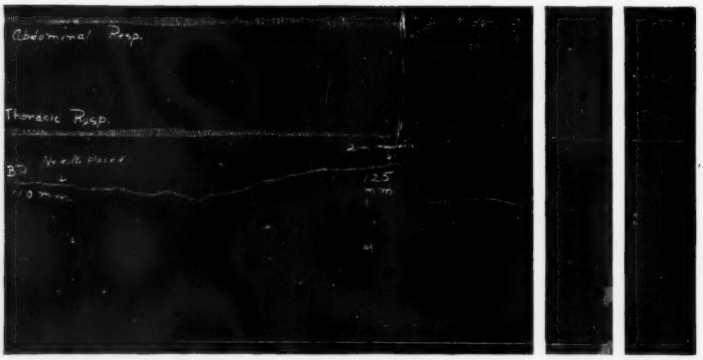

Fig. 6. The effect upon abdominal and thoracic respirations, and blood pressure of a cisternal injection of 0.12 cubic centimeter of 2 per cent novocain solution (colored with methylene blue). Dye confined to medullary region. Rabbit.

limited the fall. The average fall in systolic blood pressure in the cases not receiving preliminary ephedrine was much higher (26 per cent for those reaching to the eighth cervical segment and 48 per cent for those going above the eighth cervical segment). Whether one agrees upon the exact percentage influence of each region of the cord upon blood pressure, one must admit the cumulative action with progression upward and must expect that the higher the anæsthesia produced the greater will be the fall in blood pressure.

CHANGES IN PULSE

Table IV also shows the slowing of pulse rate during spinal anæsthesia which most observers have reported. However, this is true only in those cases in which the anæsthesia reached above the fourth thoracic segment, the average decrease in 60 such cases being eight per minute. These figures failed to give a complete idea of the degree of change since the usual increase in pulse rate due to surgical procedures and that due to the action of ephedrine had to be overcome in addition. Similar findings were noted in our animal experiments. The slowing of the pulse is due to paralysis of the preganglionic fibers (white rami communicantes) coming from the first four thoracic segments, since these fibers carry the accelerator impulses to the heart (Kuntz, 1929) and their paralysis allows the depressor vagi to act unopposed in slowing the heart.

THE EFFECT UPON RESPIRATION

Another important controversial question has been the effect of spinal anæsthesia upon respiration. That the solution in a subarachnoid injection may reach the peripheral nerves of respiration (intercostal and phrenic), the descending respiratory fibers within the cord and medulla, and even the respiratory center itself has already been shown by the dye distribution in our cadaver and animal experiments, and by the cutaneous tests in our clinical cases.

Many observers have noted a selectivity of novocain for sensory nerves, the vasomotor nerves being affected next and the somatic motor nerves last of all. Experimental proof of these facts has been offered by Kochs (1886), Santesson (1906), Gasser and Erlanger (1929). However, the margin of safety is not definite and probably not very great. The possibility of motor nerves or centers bathed by novocain solution being anæsthetized cannot be disregarded in interpreting the phenomena associated with spinal anæsthesia.

Animal experimentation. In laboratory animals in which the novocain solution was confined below the lower thoracic region (tenth or eleventh segments), there was very little effect upon the respiration (Fig. 7). When the solution reached higher levels, the intercostal nerves were progressively paralyzed, the abdominal movements increasing to compensate for the loss of costal breathing. If,

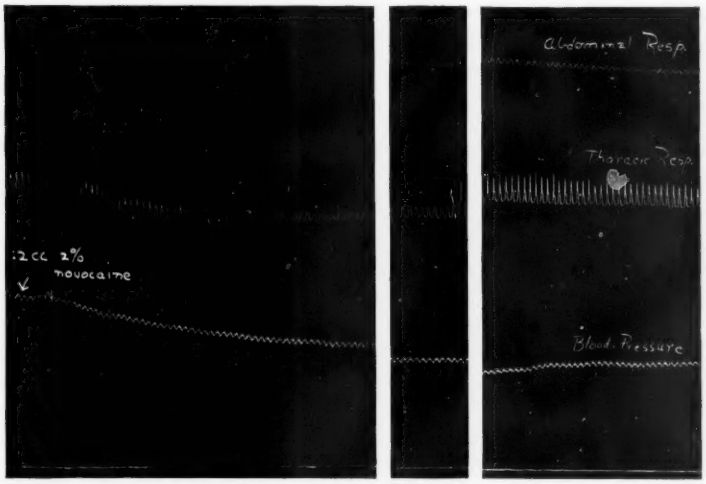

Fig. 7. The effect upon abdominal and thoracic respirations, and blood pressure of a lumbar subarachnoid injection of 0.2 cubic centimeter of 2 per cent novocain methylene blue solution which reached only to the tenth thoracic segment. Rabbit.

however, the solution continued to mount in the subarachnoid space, diaphragmatic breathing was likewise gradually depressed and finally it ceased altogether, the animal dying a few moments later (Fig. 8). Similar effects upon the respiration were noted in rats and cats by sectioning the nerve roots. When the thoracic nerve roots of both sides were sectioned, the animal continued in good condition but the character of the breathing changed to abdominal. If the cervical roots (three to eight) on both sides were then sectioned, abdominal breathing likewise stopped and the animal died. Cutting the cervical roots first (with intact thoracic roots) also caused death of the animals, diaphragmatic breathing apparently being essential to life in the cat and rat. Introduction of but a few drops of novocain solution directly into the cisterna magna caused immediate cessation of all breathing and death of the animal, from direct action on the medullary centers (Fig. 6).

These experiments show that in spite of the supposed selectivity of novocain for sensory and vasomotor nerves, the peripheral motor nerves (phrenic and intercostal) and even the respiratory center are effected by volumes sufficient to reach them and definite changes in respiration result. We have already shown that the fall in blood pressure is not dependent upon changes in respiration. Likewise the changes in respiration are not primarily dependent upon the fall in blood pressure and resulting anæmia of the brain as claimed by Labat, Koster, and others, for the character of these changes is definitely related to the progression of novocain from below upward. The changes in blood pressure and respiration are concomitant phenomena due to the action of novocain on all nerve structures which it reaches. That there is an interrelationship between blood pressure and respiration cannot be denied—a marked fall in blood pressure may cause anæmia of the brain and secondary effect upon respiration (low position of head is indicated), and complete cessation of respiration does cause a fall in blood pressure after a preliminary rise due to anoxæmia, but these are late effects and follow the primary changes due to the action of novocain itself. We are not, therefore, able to agree with Koster and others that novocain may be circulated about the cervical and medullary regions with safety, and believe that the greatest safety in spinal anæsthesia may be obtained by confining the solution below the midthoracic region and that operations above the diaphragm under spinal anæsthesia are dangerous chiefly because

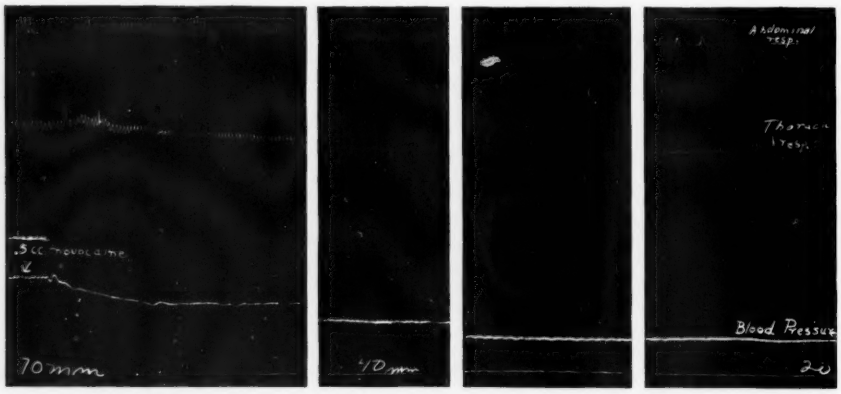

Fig. 8. The effect upon abdominal and thoracic respirations, and blood pressure of a lumbar subarachnoid injection of 0.5 cubic centimeter of 2 per cent novocain methylene blue solution which reached to the brain stem. Rabbit.

of the effect of novocain on the respiratory nerves and medullary centers.

Clinical. The following clinical case illustrates the dangers of high anæsthesia:

Mrs. S., aged 70 years, had an interposition operation for cystocele and uterine prolapse. Spinal anæsthesia was obtained by means of 300 milligrams of neocaine dissolved in 1 cubic centimeter spinal fluid and injected into first lumbar interspace. Patient was immediately placed in the dorsal lithotomy position *with the head lowered.* The blood pressure before injection was 160 systolic and 90 diastolic. Ten minutes after injection it had fallen to 80 systolic and 50 diastolic, but the patient's general condition was good and the respiration quite normal except for the fact that it was more diaphragmatic in character. Pin tests showed that cutaneous anæsthesia had reached to the first thoracic segment. Ten minutes later the blood pressure was about the same, 75/45, but the cutaneous anæsthesia had reached to the first cervical segment and the respirations were slower and more shallow. Five minutes after this, the respirations suddenly stopped, the patient became cyanotic, and the blood pressure could not be recorded. The pulse was feeble and slow and soon could not be gotten at all. The heart sounds could not be heard. Artificial respiration was immediately instituted. Strychnine sulphate grains 1/30 and alpha-lobelin grains 1/20 were given hypodermically, and adrenaline (1:1000 solution) 1 cubic centimeter was injected directly into the heart. Several minutes later the heart sounds could be made out and a feeble pulse could be felt. After about 15 minutes of artificial respiration, the patient took a few spontaneous breaths which gradually increased in rate and depth until her condition was apparently normal again.

A careful study of this case shows the typical progressive effects upon the respiratory mechanism seen in our laboratory experiments – first a change to abdominal breathing to compensate for loss of intercostals, then interference with the abdominal breathing by paralysis of the cervical segments (phrenics) and even possible effect upon the medullary centers (although this would be relatively unimportant since all the peripheral respiratory nerves were already affected). It is to be noted that there was an early drop in blood pressure (50 per cent) but that this was compatible with good respiration (though modified) and good general condition, showing the independence of changes in blood pressure and respiration. Furthermore, the blood pressure remained about the same until the respirations ceased entirely when it became imperceptible due to the anoxæmia of the medulla, or possibly to direct actions of novocain upon the vasoconstrictor as well as respiratory centers. Until that time, however, the blood pressure had reached its maximum fall due to peripheral vasodilatation and this correlated well with the level of cutaneous anæsthesia to and above the first thoracic segment. That the head was already down when the respiratory difficulties began gives further evidence that the latter were not due to anæmia of the medulla resulting from fall in blood pressure. It is interesting to note that although there

was some fixation of novocain early, this was only partial and the effect continued to spread upward for 20 to 25 minutes after injection. It has been our observation that in older patients with sclerotic vessels and high blood pressure, the fall in pressure is apt to be sharper and more intense than in younger patients with elastic vessels and lower blood pressure. This does not contra-indicate the use of spinal anæsthesia in older patients but necessitates the use of greater caution and the restriction of the anæsthesia to lower levels. The recovery of this patient must be ascribed to the artificial respiration instituted, since the essential cause of the reaction was respiratory failure and restoration of breathing was all important for recovery. However, the use of stimulants may have had some additional favorable effect, adrenaline particularly stimulating the waning circulation.

EFFECT UPON INTESTINAL MOVEMENTS AND TONE

It has long been observed that spinal anæsthesia gives the greatest amount of relaxation in abdominal operations. This is no doubt due in part to the great muscular relaxation of the abdominal wall and to the absence of violent respiratory movements, but the condition of the intestine itself is of very great importance. It has been noted that the intestine is usually collapsed and free of distention, a condition which has been of the greatest advantage in difficult surgical procedures. The basis for this condition has not been so clear, though generally regarded as due to paralysis of the preganglionic sympathetic fibers in the roots of the fifth to the twelfth thoracic spinal nerves (splanchnics). Ochsner and Gage (1930) have shown a temporary cessation which is followed by hyperactive movements and increased tone after cutting the splanchnics. They have also found a similar effect from spinal anæsthesia reaching about the origin of the splanchnics.

Pithing the cord in our experimental rats was followed by a marked increase in the rate and amplitude of intestinal movements as well as a general increase in tone. The following are protocols of typical rabbit experiments illustrating these points.

Fig. 9. The effect upon intestinal movements and tone of a lumbar subarachnoid injection of 0.15 cubic centimeter of 2 per cent novocain methylene blue solution. Dye reached to fourth thoracic segment. Rabbit.

Rabbit 17. Weight 2 kilograms. Anæsthesia, 2 grams urethane. A soft rubber catheter was inserted into the rectum for a distance of about 6 inches, fixed with a purse string suture, and sealed with collodion. The free end was connected with an air tambour, the lever arm of which was made to write upon a kymograph. After a record of the normal intestinal movements and tone was obtained, 0.15 cubic centimeter of 2 per cent novocain colored with methylene blue was injected into the subarachnoid space through an upper lumbar laminectomy wound. After a very short latent period, there was a marked increase in the frequency and intensity of the intestinal movements with a general increase in tone (Fig. 9). At postmortem, the anæsthetic solution was found to have reached to the fourth thoracic segment.

Rabbit 19. Weight to 2.85 kilograms. Anæsthesia, 2.8 grams urethane. Artificial respiration. Tambour was arranged as in preceding experiment. Both splanchnics were isolated just below the diaphragm. After a record of normal intestinal movements and tone was obtained, the left splanchnic was cut. This was followed by very little change in the record but, when the right splanchnic was severed, there was an almost immediate increase in frequency and intensity of movements as well as tone. Injection of 0.2 cubic centimeter of 2 per cent novocain colored with methylene blue into the subarachnoid space was followed by a still greater increase, especially in intensity of contractions (Fig. 10). Postmortem examination showed that the novocain solution had reached to the sixth thoracic segment.

These experiments show that spinal anæsthesia affects intestinal movements in the same way as cutting the splanchnics and that the effects must be due to paralysis of the preganglionic fibers in the nerve roots of the fifth to twelfth thoracic segments from which the splanchnics take origin. The additional effect produced by spinal anæsthesia after the splanchnics are sectioned is due to the fact that the section of the latter is of necessity

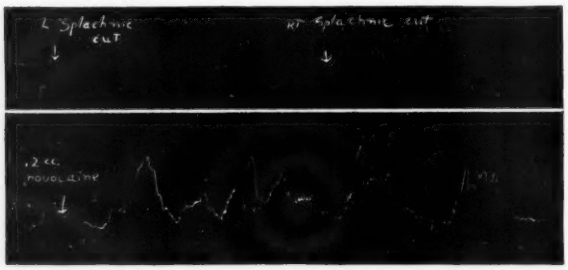

Fig. 10. Tracing showing the effect upon intestinal movements and tone of cutting the left and right splanchnics, respectively, followed by a lumbar subarachnoid injection of 0.2 cubic centimeter of 2 per cent novocain methylene blue solution. Dye reached to sixth thoracic segment. Rabbit.

incomplete whereas spinal anæsthesia catches all the fibers below its upper level. Because of this action on the intestinal tract, spinal anæsthesia is not only advantageous for abdominal operations but may be used in the treatment of paralytic ileus. This method has often been successful after other measures have failed to control abdominal distention —especially postoperative distention.

SUMMARY AND CONCLUSIONS

1. Subarachnoid injection of novocain dissolved in spinal fluid produces a convenient and satisfactory anæsthesia for operations of the abdomen, perineum, and lower extremities.

2. The height of anæsthesia is dependent upon the volume and concentration of the solution, the rate of injection, the position of the patient and, to a lesser degree, the site of injection.

3. No definite levels for specific volumes and concentrations will hold for all patients but, by varying these two factors, the tendencies of spread can be anticipated.

4. In general, the use of 150 milligrams of novocain dissolved in 1 cubic centimeter of spinal fluid for work in the lower abdomen, perineum, and lower extremities, and 200 milligrams in 1 to 2 cubic centimeters for work in the upper abdomen are most satisfactory. The duration of anæsthesia is directly proportional to the concentration and inversely proportional to the spread.

5. The fall in blood pressure is dependent upon paralysis of the vasoconstrictor nerves or center, and not upon respiratory depression — at least not until the latter has ceased entirely. Adrenaline and ephedrine, acting upon sympathetic myoneural junctions, are effective means of preventing and combating this fall in pressure. Keeping the head lowered during anæsthesia is important in maintaining the blood supply to the medulla when the pressure is low, but is only of secondary importance as far as changes in respiration are concerned.

6. The cause of death following spinal anæsthesia is primarily respiratory, due to action upon the peripheral respiratory nerves or upon the medullary center. The most effective treatment for respiratory failure is artificial respiration, although drug stimulants may be of some value.

7. Spinal anæsthesia is a safe anæsthesia when kept below the level of the mid dorsal region.

8. Spinal anæsthesia causes an increase in intestinal movements and tone due to paralysis of the preganglionic sympathetic fibers (splanchnics) which carry the majority of inhibitory impulses to the intestine. This fact may be used clinically in the treatment of paralytic ileus.

We wish to take this opportunity of expressing our appreciation to Mr. Ivan D. Farnsworth and Mr. Howard Black, College of Medicine, University of Nebraska, for their valuable technical assistance in this work.

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FACTORS WHICH DECREASE RISK IN OPERATIONS ON COLON AND RECTUM

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IN the treatment of carcinoma of the colon and rectum, one inevitably finds that the condition unfortunately is not selective only among persons best able to undergo a major surgical procedure, but that the selection is indiscriminate. Suffice to say that at least one factor in a low mortality rate is directly dependent, not on a well defined standardized technique for operating on growths in certain situations, but on the ability and judgment of the surgeon to determine the patient's condition and be guided by it to select the type of procedure best suited for the particular case.

In recent years operations on the colon and rectum, although still fraught with many undesirable features, have been accomplished with a degree of proficiency which materially enhances the prognosis in such cases. The operative mortality as well as the morbidity, has been reduced materially. However, the very nature of the pathological changes, usually malignant, encountered in these cases, and the site of the lesion in a heavily contaminated and infected bowel, precludes the possibility of obtaining results comparable with those obtained in the upper part of the gastro-intestinal tract where the bacterial content is less profuse and many of the lesions are benign. Many years ago Cushing demonstrated that the bacterial content became greater in the more distal portion of the intestinal tract, and reached its maximum in the terminal portion of the ileum. If one considers only malignant conditions of the upper and lower parts of the alimentary tract, the percentage of operability and end-results of carcinoma of the colon and rectum do not suffer by comparison with neoplasms of the stomach and small intestine, although this may not be equally true of immediate operative mortality.

Many individual factors have contributed to the increased safety of present day treatment of colonic and rectal lesions. A few of the more important factors may be divided into three groups: pre-operative, operative, and postoperative.

PRE-OPERATIVE FACTORS

In cases in which the symptoms point to a possible lesion in the colon or rectum, it should be remembered that the administration of barium by mouth makes a serious condition more serious. Lesions of the large intestine and rectum rarely cause complete obstruction, particularly those of a malignant nature. However, partial obstruction is almost invariably present; in fact it is usually because of this mechanical interference with the motor mechanism that symptoms develop. If barium is administered orally in such cases, as is often done, complete obstruction may be produced, the very thing one strives to overcome. Figure 1 is illustrative of such a case. The lesion was in the descending colon. Barium was given by mouth and complete obstruction occurred soon afterward. Enterostomy was necessary to save the patient's life.

An aerogram affords superior visualization of the colon, and if a lesion is found to be present, obstruction may be obviated. In making the aerogram, an enema of thin barium is given and the patient is allowed to expel it immediately as thoroughly as possible. Then, by means of a small bulb syringe, air is introduced into the rectum until the colon is slightly distended. The procedure is carried out under direct fluoroscopic visualization; it will be found that sufficient barium will adhere to the mucosa of the colon so that a lesion, if present, is easily discernible.

If a lesion of the colon or rectum is suspected, a careful digital examination should always be made. All lesions of the rectum and many of the lower part of the sigmoid can be felt. Occasionally a lesion of the middle of the sigmoid becomes intussuscepted and can

be felt by the examining finger. Proctoscopic examination serves to identify lesions of the lower part of the bowel within 24 to 28 centimeters from the anal margin, and their gross appearance in most instances makes it possible, as a rule, to determine their nature and extent. A specimen should be removed on proctoscopic examination for microscopic study, because in many instances the grade of malignancy with other factors may be of value in determining the type of surgical procedure to be carried out. The roentgenogram furnishes tangible information concerning the situation and nature of lesions higher in the rectum.

To facilitate transfusion, should it be necessary, typing of blood should be done in all cases in which surgical lesions of the bowel are present.

After the diagnosis is established, 3 to 5 days' hospitalization prior to operation is extremely desirable. During this time particular attention is given to the general condition of the patient and to the proper preparation of the abdomen and colon. A diet high in carbohydrates and low in residue is recommended. An effort is made to establish a reserve of carbohydrate energy which will be readily available for the first few days following operation. A generous supply of fluids is likewise advisable. Frequently, if pronounced anæmia exists, especially in the presence of a growth in the right half of the colon, one or more transfusions greatly increase the general forces of resistance.

Vaccine (streptococci and colon bacilli) is administered 3 days before operation as a routine, thus permitting the proper interval for the optimal extent of mobilization of the peritoneal defensive forces prior to operation. In preparing the bowel itself, an initial mild cathartic, that is, citrate of magnesia, may be advisable in some cases. During the 48 hours prior to operation, a low residue diet, and warm enemas, twice daily, of physiologic saline solution aid in emptying and cleansing the bowel on the morning of the operation. Generous doses of paregoric may be given during the first 12 of the last 24 hours before operation. In event the saline enemas are inadequately expelled, the colon is aspirated in the morning before operation. Care should be taken to have patients void immediately before operation. A filled urinary bladder does not increase the visibility in operating in the pelvis.

OPERATIVE FACTORS

There are numerous and diverse factors, from an operative viewpoint, which may contribute to the safety of the patient suffering from a growth in the colon. These factors may be divided into two main groups: those involving surgical judgment, and those concerned primarily with surgical technique.

In the first group, the most important factor is the choice of operation; this is of particular significance in cases of lesions in the left side of the colon and in the rectum. What might be the ideal operation for a robust patient of middle age would likely prove too strenuous for one less sturdy and of more advanced years. As a rule, a fine balance must be drawn between the desire to perform a radical operation which offers the best ultimate prognosis, and a less extensive operation with a lower initial mortality and less chance for cure. The experience and ability of the surgeon and his familiarity with this field should influence the decision.

In dealing with lesions of the rectum and rectosigmoid, the choice of operation ordinarily lies between five different procedures: local excision, posterior resection with a sacral anus, colostomy and posterior resection (one or two stages), combined abdominoperineal resection in two stages, or combined abdominoperineal resection in one stage, as advocated by Miles. The main points to be considered in selecting the proper operation for the individual patient are: age, general condition, grade of malignancy, local extent of the growth, and the experience and ability of the surgeon. In cases in which patients are particularly debilitated and of advanced years, and the grade of malignancy is comparatively low (1 or 2) treatment by radium and roentgen rays may be carried out. If after 6 or 8 months examination reveals a marked decrease in the size of the lesion, a surgical procedure which enables the continuity of the bowel to be re-established may be attempted.

The more radical procedures which favor cure may be employed as a routine in a given case unless contra-indicated. Malignant lesions graded 4, fortunately uncommon in this situation, are not suitable for operation; however, the surgeon is loath not to attempt radical removal because occasionally such removal may afford the patient many additional years of comfort.

In cases of growths of the descending colon and sigmoid, the choice of operation is usually between obstructive resection, a Mikulicz operation, or some type of modified anterior resection. Primary resection with end-to-end anastomosis is prohibited by a high operative mortality. The Mikulicz operation, although perhaps the safest procedure from the point of view of immediate recovery, may not entail widespread removal of the node bearing area, and may be followed by local recurrence. However, many patients are alive 10 years or more following resection by this method. Resection also affords by far the most desirable palliative procedure if it is performed in the face of extension beyond the site of operation. For lesions of the transverse and descending colon, obstructive resection, as described by Rankin, is deserving of consideration. An accompanying cæcostomy is often of value. Carcinoma of the cæcum or ascending colon is best removed by ileocolostomy, either as a preliminary procedure to, or associated with, resection of the right half of the colon. The two stage procedure in most cases is probably the wiser. If death occurs following a one stage resection, one can always realize, in retrospect, that a procedure of less magnitude could have been adopted.

The selection of an anæsthetic agent is ever a moot question, and to some extent is dependent on the facilities immediately available. For perineal work, a sacral block is practically ideal. If the peritoneum is opened, the patient will experience some discomfort for which a small amount of gas may be administered. Spinal anæsthesia has the advantage of affording complete relaxation and ablation of peristalsis for several hours subsequent to operation. If there is too much traction on the mesentery, nausea or straining will occur, which to some extent overcomes these advantages. This anæsthesia is considered advisable in the presence of pulmonary complication or when operating on excessively obese patients. It is less suited to patients with marked hypertension, arteriosclerosis, extreme nervous instability, or to those of advanced years. If spinal analgesia is contemplated, proper preliminary medication by mouth is particularly advantageous. We use pentobarbital sodium, grains 1½ (0.1 gram) the night preceding operation and a similar amount one hour before operation, supplemented by morphine grain ¼ (0.01 gram) with atropine sulphate grain 1/150 (0.0004 gram). A properly administered inhalation anæsthesia with induction of nitrous oxide or ethylene and ether for maintenance still remains difficult to excel in many cases. As with most variables in surgery, it is unwise to permit the use of a given anæsthetic as a routine for a certain operation, and thereby individualize a technical procedure in preference to individualizing the patient.

The general plan of resecting the colon and rectum in two and occasionally in more stages, in contrast with primary resection, has received and has merited considerable attention. The advantages of this plan, especially if operation is performed on a debilitated patient, are numerous. The shock and severity of the initial operation are greatly reduced. At the time of the second operation the patient has, to some extent, vaccinated himself against subsequent insults of peritoneal contamination. Many times, a growth densely adherent and fixed when first encountered will become sufficiently mobile during the ensuing few weeks, because of recession of surrounding inflammatory processes, to permit resection without widespread dissection and trauma. The patient is not suffering from the effects of a long standing, partially obstructing lesion and is therefore in the best possible condition at the time resection is performed. The time between the stages of operation may be varied according to the general condition and age of the patient and also the smoothness of convalescence following the initial operation. A patient whose general health is good, and who recovers rapidly following the primary surgical procedure, which is usu-

ally decompression in one form or another, may well be kept in the hospital for 3 to 4 weeks, when the second stage may be performed safely. If patients are of advanced age perhaps with complications during convalescence from the first stage such as a pulmonary infarct, phlebitis, or parotitis, they should be allowed a more prolonged (4 to 6 weeks) period of rest before resection is undertaken. On the other hand, in our enthusiasm for operations in stages, a number of comparatively young and sturdy patients are probably denied the privilege of primary resection, with the resultant advantages of immediate removal of a malignant growth and shortened stay in hospital. Here again, surgical judgment and consideration of patients individually should be paramount. Occasionally we find unexpected extension of a malignant growth at the second operation, even if only a few weeks have elapsed. This is unusual, and is most prone to happen among young persons or those who harbor a highly malignant neoplasm. Initial extirpation in such cases is most desirable and usually feasible. If, for any reason, the growth is not primarily removed, its ablation should be postponed no longer than is absolutely necessary.

Various problems arise at the time of operation which demand careful consideration and judgment on the part of the surgeon if the patient is to have the benefit of all possible factors of safety. Prominently, in this regard, arises the desire to perform some type of obstructive operation for lesions of the distal half of the colon in the presence of distinct evidence of chronic or even subacute obstruction. When acute obstruction exists, there is no question but that immediate and simple decompression should be the procedure of choice. Even when the obstruction is chronic, primary resection, although it may appear desirable, should not be performed. In the presence of a thick walled and somewhat dilated colon, thought should be given to drainage and physiologic rest for the involved segment, with all thought of resection postponed to a subsequent date. It is often a great temptation to overlook this consideration when one encounters a small, encircling, freely movable growth, but strict adherence to this plan will usually afford far superior results. A Mikulicz operation can often be carried out safely under such conditions; however, the indications for this operation are limited.

Cæcostomy is often a life-saving procedure. "Blind cæcostomy" finds its greatest field of usefulness in the relief of acute obstruction of the large bowel, of unknown site and etiology. If the portion of the cæcum brought out of the abdomen is walled off from the abdominal incision by means of vaseline gauze, it may be punctured in 6 or 8 hours with safety and the critically ill patient can be restored to comparative health preparatory to adequate observation, and subsequent surgical procedures can be directed toward cure. Acute obstruction, if the patient is of advanced years, developing suddenly, is usually caused by a carcinoma of the colon distal to the hepatic flexure. The obstruction may not be complete and may be partially alleviated by gentle irrigations. In certain other cases, usually in association with more or less extensive surgical procedures on the distal half of the colon, the estab ishment of a safety valve, in the form of cæcostomy, materially reduces the risk of operation. If there is no such opening in the cæcum, occasionally postoperative ileus or temporary obstruction will develop, and, if it is not relieved, will result in such marked dilatation of the bowel that the intestinal musculature apparently becomes paralyzed and unable to restore the bowel to normal, although the original etiological factor for the dilatation is removed later. Likewise, when the right half of the colon is resected in a one stage operation and ileocolostomy is performed, it is wise to establish temporary ileostomy at the same time, otherwise it may become necessary as an emergency procedure under less desirable conditions.

The advisability of draining the operative site following various surgical procedures on the colon may usually be decided without difficulty, according to certain general surgical procedures. When large pockets are left behind, even after complete peritonization, as in resection of the right side of the colon, it is wise to insert a small soft rubber tube to permit subsequent discharge of collections of

serous material. Likewise, if there has been gross contamination, drainage of the involved area is most desirable. Vaginal drainage may sometimes be carried out to advantage; for example, in cases of total colectomy. The drainage of posterior wounds following excision of the rectum can well be reduced to a minimum, as large packs of gauze left in place for several days merely prevent adequate drainage and promote local absorption of toxic material. If these wounds are properly cared for after operation, we need not fear collections of toxic material in pockets which are so frequently caused by local areas of rapid healing.

In the second group are numerous factors pertaining to actual operative technique which tend materially to reduce the hazard of surgery. Although technical maneuvers vary in details with each individual operator, certain general principles are equally applicable in every surgical attack on the large bowel. Prominent among these principles is the policy of reducing to a minimum the handling of any growths of the colon. All tumors of the colon are heavily infected, and the organisms and products of inflammation are not limited in their extent to the mucosa, but tend to invade the wall of the bowel and adjacent lymph channels. Handling or trauma of any description promotes direct contamination of the serosa and surrounding peritoneum, even though the bowel is not actually torn. It is partially for this reason that in performing combined abdominoperineal resection for carcinoma of the rectum or rectosigmoid, many surgeons consider it advisable first to mobilize the rectum posteriorly, encase it in a sterile rubber glove, and then open the abdomen to complete the operation. By performing resection in this sequence, mobilization of the highly infected portion of the bowel in which the growth is situated, is all done extraperitoneally. If the growth is intra-abdominal, all exploration should be done first, and then the operative site should be packed off from the remainder of the abdomen; last of all, attention should be directed to the growth itself.

Before mobilization of any lesion of the rectosigmoid or sigmoid, the left ureter should be identified. Although frequently this may seem superfluous, nevertheless as the dissection is continued, even the most experienced surgeon may hesitate just in time or a little too late to avoid this structure. Only rarely does the right ureter become involved in the region of growths in the colon; however, when the pelvis is filled with a densely adherent mass, accurate localization of the right ureter is not a wasted gesture.

Great emphasis has always been placed on the paucity of the blood supply to the colon. The importance of maintaining adequate circulation in any surgical procedure on the large bowel has been stressed frequently, and many calamities have been credited to failure to observe this point. The recent excellent work of Steward has shown that the arteries to the colon are far more constant than was previously supposed, although there may be some variation in the course of the branches. One simple rule suffices to obviate most difficulty in this regard. After the observance of ordinary anatomical facts, one need merely determine, by close observation, the presence or absence of pulsations in the vessels supplying the segment of bowel in question. These pulsations are readily seen, if they are present, and afford positive proof of arterial circulation in the region where they are visualized. If they cannot be found, further resection is generally indicated.

Accurate peritonization of all raw surfaces is important; it is, of course, a cardinal surgical principle, regardless of the intra-abdominal viscus to be attacked. It is of particular importance in the formation of a new peritoneum of the floor of the pelvis after the completion of a combined abdominoperineal resection, as weakness in this diaphragm may be most disastrous. Accurate peritonization is also essential following resection of a lesion in the right side of the colon or in the sigmoid.

POSTOPERATIVE FACTORS

Patients subjected to surgical procedures on the colon are prone to have, as a group, a more disturbed postoperative course than those on whom the average laparotomy is performed, and for this reason they require more careful postoperative supervision. Here again, adherence to certain general principles

Fig. 1. Obstruction caused by the administration of barium orally.

affords the greatest number of satisfactory results.

During the first few days, the abdomen must be kept quiet. To accomplish this, as well as to relieve pain, morphine may be used liberally for the first 48 hours. It is usually safer not to give fluids by mouth until gas is expelled from the bowel. The administration of small quantities of water by mouth is begun usually 48 to 60 hours after operation. When the patient is taking nothing by mouth, and even after fluids have been started cautiously, the mouth should be kept moist and clean as an aid in the prevention of parotitis, which seems especially prone to affect patients subjected to operations on the colon. During this interim the fluid balance is maintained by means of hypodermoclysis of physiologic sodium chloride solution and occasionally with intravenous injections of a 5 or 10 per cent solution of glucose. The fluid should be administered intravenously, cautiously and slowly; one liter of saline solution should require 1 hour, saline and glucose solution 2 hours, and given under the direct supervision of a physician. In recent years, the popularity of intravenous injections has increased tremendously but not without dire, as well as beneficial, results. The use of intravenous solutions as a routine method of supplying an adequate intake of fluids seems inadvisable. The ease with which these solutions are prepared and administered has augmented their popularity, and although they are a great boon in many cases, their promiscuous use may cause undesirable effects. They are more likely to provoke unfavorable reactions if patients are obese, have short necks, hypertension and arteriosclerosis, weakened myocardium, or in the presence of considerable infection. If there has been evidence of phlebitis, thrombosis, or pulmonary infarcts, solutions should not be given intravenously. The ideal solutions have been found to be physiologic saline, 5 or 10 per cent glucose, and 5 per cent glucose in physiologic saline.

Occasionally, when the patient's resistance has been lowered by a few difficult postoperative days, transfusion of blood will often effect a surprising turn for the better. This may be repeated as indicated.

In the treatment of ileus, hot stupes, pituitary extract or physostigmine or, at times, acetylcholine have proved of some benefit. Most patients respond to this treatment, although sometimes enterostomy, even in the absence of actual mechanical obstruction, is indicated and affords satisfactory results. The intraspinal injection of procaine has in many instances been dramatic in its relief of ileus. It should be administered, however, before process has reached stage producing shock.

The etiological factor of parotitis which so frequently develops following operations on the colon, remains undetermined, although the treatment of the condition by the early application of radium is most satisfactory. Occasional repeated applications are necessary, but after the proper amount has been administered the swelling disappears and suppuration does not occur. Rarely the same type of inflammatory process is seen in the submaxillary gland. Pulmonary, cardiac, urinary, and other complications, when they occur, are treated much the same as they are following any type of surgical procedure.

SUMMARY

Careful pre-operative study and adequate preliminary preparation of the colon, abdomen, and general metabolic reserves en-

hance the surgical prognosis in cases of malignant lesion of the large bowel. The consideration of patients individually, which entails the judicial selection of operative procedure and the proper technique for its consummation, is an essential prerequisite for satisfactory results. Intelligent postoperative care, based on an accurate appreciation of the altered pathological physiology, adds further to the patient's safety.

STUDIES ON THE IMMOBILIZATION OF THE NORMAL JOINTS[1]

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WITH the advent of the modern treatment of fractures, much has been written concerning the prompt restoration of motion and ensuing lack of joint limitation resulting from the early mobilization of joints. This has been contrasted with stiffness and loss of motion frequently found in those limbs which have been fixed for a long period in a rigid dressing, such as plaster of paris, and the advocates of early joint motion have declared that disability periods can be shortened, function can be more quickly restored, and permanent disability can be lessened if their recommendations are followed. Clay Ray Murray, of New York, has written extensively on this subject and states that in his cases, treated with early motion, the period of disability was materially shortened.

Although a great deal of opinion has been voiced as a result of clinical observation, very little experimental work has been done of recent years to verify or disprove these observations. On the other hand, there are still quite a few men of prominence who believe that immobilization of a normal joint has little effect upon its structure and that the above mentioned procedures frequently do a great deal of harm and very little good. They further claim that when stiffness of joints does result, it is because the joints that are immobilized are not normal in character, but are suffering from some pathological condition. It is asserted by many that prolonged immobilization of joints, already the seat of inflammation, will tend to cause changes that are difficult subsequently to eradicate. This assertion seems to be based solely on clinical observation. It is the only hope of function in some cases. In favor of the lack of damage by immobilization, its advocate brings forth the fact that the joints of children can be immobilized for weeks or months and when released from apparatus rapidly return to normal function without disability.

Because of this confusion and of paucity of actual experimental work done upon this subject, we have made an effort to review and check the work that has been done and to determine, if possible, just what pathological changes may occur in the experimental animal following immobilization.

Carl Reuber, in 1874, showed in experimental animals that certain definite changes occurred from prolonged inactivity. He noted that in the cartilage beyond the surfaces that are habitually in contact, large round cartilage cells passed by gradual acquisition of cell processes into spindle shaped or stellate connective tissue corpuscles of the synovial membrane. In 1909, P. W. Nathan, speaking of joint cartilage, stated that it is a resistant, inert substance which stands a marked amount of irritation without noticeable change in structure; when destroyed it does not regenerate. It does not exhibit inflammatory change. Proliferation is only in response to function. In 1916, Ely and Cowan, in experimental work on animals, reported similar findings. Nathan further notes that the normal layer of vascular connective tissue at the periphery of the joint where surfaces are not in contact increases when there is loss of function and decreases when there is an increase in function.

[1]Presented before the San Francisco County Medical Society, November 15, 1932.

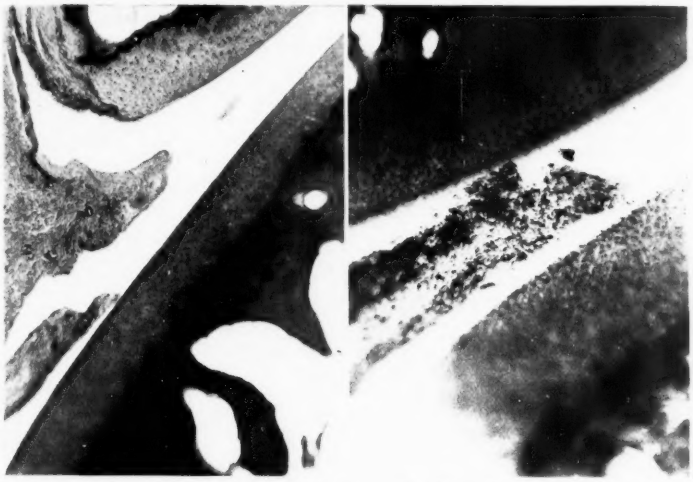

Fig. 1, left. Periphery of joint showing normal proliferation of the areolar tissue. Note the relative width of the joint space and the smooth outline of the articular cartilage.

Fig. 2. Dog 3. The encroachment between the articular surfaces. Note the roughening and changes in the hyaline cartilage of the opposing joint surfaces.

The last authentic work to be done on this subject is that by Walther Muller in 1923, who made the following observations: Only extreme grades of fixation give apparent discernible changes and then only after a long period of immobilization. The limitation of joint motion is first caused by shrinkage of the adjacent muscles and the capsular structures. In extreme grades of immobilization by means of skeletal traction and fixation of plaster of paris, we find an apparent ingrowth of the connective tissues from the periphery of the joint, growing to the point where the articular surfaces are in apposition. This is similar to a condition which takes place in joints where an old dislocation has been unreduced. He also noted some thinning of the joint cartilage with a tendency toward necrosis and fibrous degeneration.

OUTLINE OF EXPERIMENTS

For our purpose, four adult dogs were selected and under anæsthesia their right hind legs were immobilized in plaster of paris, from the toes to above the knee joint. These plaster dressings were retained for a period of 1 to 3 months. The animals were then sacrificed and their ankle joints examined, both microscopically and mascroscopically. In order further to control the situation and give us a basis of comparison, a normal joint was taken from a dog which was of similar age, healthy, had not been subjugated to this enforced inactivity.

In one or two cases the plaster was either lost or destroyed by the animal on several occasions, but promptly reapplied before any effort had been made to utilize the joint under observation. We do not believe that these complications had much effect upon the ultimate findings, as under the microscope, all specimens showed certain definite variations from normal.

Observation on removal of the plaster in some cases showed some apparent soft tissue swelling about the joint, but no attempt at eliciting the range of motion was made, in order not to disturb any changes that might have occurred which could be subsequently identified under the microscope.

Dog 1. February 24, 1930. A large, brindle colored male, half wolf and half police dog, was given morphine sulphate and anæsthetized with ether, and the right hind leg from foot to knee was put up in

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 3. High power photomicrograph showing the thinning irregularity and fibrillation of the joint surfaces following immobilization.

Fig. 4. Dog 1. Elimination of the joint space with substitution of fibrous tissue and fibrous cartilage for hyaline cells.

Fig. 5. High power photomicrograph showing close approximation of the surfaces of the joint with destruction of the cartilage and replacement of the fibrous tissue.

plaster of paris. Anæsthesia was given merely for the purpose of having the dog quiet until the cast had been set. March 8, 1930, lost plaster dressing in afternoon. March 13, 1930, plaster was renewed. March 31, 1930, plaster came off. April 1, 1930, plaster was renewed. The knee was freely movable. April 18, 1930, the plaster was renewed. The old one was partly chewed off and there was some swelling just above the ends of the casts. April 22, 1930, dog lost the plaster. It was renewed after 4 hours, and reinforced with tin lining. There was some rawness on leg underneath cast. Aristol powder was applied. May 10, 1930, dog lost plaster again; it was renewed early in the morning; and he chewed it off again next day. May 12, 1930, dog was killed with gas. No swelling was noticed.

Dog 2. March 3, 1930, a white adult male dog, terrier type, was anæsthetized with ether and a plaster-of-paris cast was applied to the right ankle. May 29, 1930, the dog was killed with chloroform. No swelling was present in the right leg but the left leg showed considerable œdema from the knee down. The swelling was due to bite in groin by other dogs with resultant interference with the blood supply. The leg showed great extravasation of blood.

Dog 3. March 3, 1930. A tan brown female, still young, had a plaster-of-paris cast applied to right ankle, under ether anæsthesia. April 14, 1930, the dog lost cast, which was renewed at 1 p.m. There was some swelling around the ankle and up to 2 inches below the knee. The cast was lost again the same evening. April 15, 1930, cast was renewed at 8:30 a.m. April 24, 1930, dog lost cast at noon. There was some swelling around the joint. New cast was applied at 5 p.m. April 29, 1930, dog lost cast and was killed with chloroform. The knee was slightly swollen.

Dog 4. March 13, 1930. A large, long haired, black and white male dog had a cast applied to the right hind leg at the ankle, just as in previous animals. April 14, 1930, dog died. This cast was still intact. At postmortem examination no gross changes were noted on inspection.

After fixation and decalcification, sections were made of the entire ankle joint, showing both the talocrural and the talocalcaneal articulations stained with hæmatoxylin and eosin. Study of these in all the animals disclosed the following findings. The articular cartilages seem to be closer approximated; the surface layer of the cartilage was definitely roughened and shaggy. The thickness of the cartilage was decreased at the point where the greatest pressure of the apposing articular surfaces occurred. In places between the articular surfaces were found isolated irregular bands of fibrous tissue, containing only a few cells, and in places were masses of homogenous staining

material with the characteristics of fibrin. This was more marked in the case of Dog 1. These were apparently tags of synovial membrane with fibrin formation, for further section showed an apparent continuity of this tissue with some of the articular cartilaginous surfaces, the edges of which had become irregular. The cartilage matrix was degenerated, with a definite decrease in the number of hyaline cartilage cells present. Here there was a substitution of a stellate type of cell resembling the connective tissue variety. Adjacent to these areas were definite points of vacuolization, usually close to the peripheral margin. The remaining cartilage, while of apparently normal cell content, stained noticeably deeper, as though increased calcification was present. This was not primarily a decrease in calcification in the degenerated areas, as the normal tissue which was put through the same staining process was noticeably lighter in contrast. At the periphery of the joint a marked change was apparent, with an increase in the amount and density of the fibrous tissue. This had definitely invaded the cartilaginous surface with a condensation of the hyaline matrix and a loss of the hyaline type of cell. A structure resembling fibrous tissue was found to replace the normal cartilage, forming a cell of the stellate variety, in some places invading the cartilage to a depth of 25 per cent of its thickness. Occasionally this tissue had penetrated the cartilage and could be seen invading the subcartilaginous zone. The replacement cells were of a closely woven stellate type in parallel bands, blending with the tissue normally found on the periphery which is of the loose areolar type. In some sections this infiltration of fibrous tissue extended for a distance of ¼ of the low power field into the joint itself from the periphery.

In the subchondral zone the bone trabeculæ were not sharply defined, and in places the marrow spaces were filled with large numbers of red blood cells. In one case, however, in which the dog was considerably younger than the others, there was distinct evidence of lymphoid infiltration. This fact brings up the question as to whether the change in the joint caused by immobilization differs in young and older animals. We hope to investigate this matter later.

CONCLUSIONS

Definite microscopic anatomical changes were noted as follows:

1. The cartilage shows a closer approximation of articular surfaces.
2. There is thinning, irregularity, fibrillation, and in places vacuolization of the cartilage.
3. Fibrin plaques are evident between the joint surfaces apparently related to the synovial membrane.
4. The areolar tissue about the periphery of the joint becomes of a denser consistency and encroaches between the articular surfaces in places, causing a substitution of the cartilage with connective tissue type of cell.
5. No definite fibrous adhesions are demonstrable between the joint surfaces.

The results of this work have confirmed, in our opinion, that the conclusions of previous investigators, who have found definite anatomical changes in joints as the result of immobilization, are correct. The findings of both Reuber and Nathan were present with striking regularity in all sections.

We cannot entirely agree with Muller, who, while confirming the anatomical observations of earlier investigators, stated that only extreme grades of immobilization give discernible changes. In our series, the immobilization was not complete, as if the bone had been in plaster of paris with skeletal traction, nor was the immobilization constant during the entire period. However, in spite of the absence of these factors, the fixation was complete enough to give the microscopic picture above described.

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THE EFFECT OF ANTERIOR HYPOPHYSIS ON CONCEPTION AND PREGNANCY IN THE GUINEA PIG[1]

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THIS investigation was undertaken somewhat as a corollary to one previously reported concerning the effect of injections of estrin on conception and pregnancy in the guinea pig (6). Since it had been found that estrin in small doses prevented conception and in considerably larger doses terminated pregnancy, it was considered possible that stimulation of the follicular apparatus of the ovary might result in the production of sufficient estrin to bring about the same results.

The possibility was suggested by the well known work of Smith and Engle, and of Zondek and Aschheim, on the relation of the anterior hypophysis to the gonads. After this work was undertaken, a report by Evans and Simpson was seen, which stated that "occasionally premature birth resulted from the stimulated production of folliculin." Engle and Mermod reported that transplants of anterior hypophysis in rats and mice terminated pregnancy uniformly in the first two-thirds and often in the last third. They thought sufficient dosage would do so in this stage also.

Subsequently followed efforts to differentiate the hypophyseal hormones, and the opinion became general that there are two such principal hormones affecting the ovary, one a follicle-stimulating and the other a luteinizing hormone. These separated hormones were not available for this investigation, though an extract from the urine of pregnant women was supplied by a manufacturer.[2] There is no doubt that both hormones were present in this extract, though the luteinizing element attracted more attention, due to its terminal effect on the ovaries of the test animals.

In this connection it is interesting to note that Lepine determined the co-existence of both antagonistic hormones and found that one brought about follicular maturation of the adolescent female and abortion of the pregnant female, while the other caused the formation of hæmorrhagic follicles and the yellow bodies. Werchratsky reported that injections of a hormone extract from the urine of women in the second period of pregnancy into pregnant animals resulted in abortion. Gastimirovic reports ovulation produced by the luteinizing hormone (Prolan B).

In addition to the urine extract, freshly expressed juice of anterior lobes of bovine hypophyses was used. The glands were obtained from the slaughterhouse and the injections made within less than 2 hours after the animals were killed. The capsules of the glands were stripped off and the organs laid in half in the mid-saggital line. The anterior lobe was separated from the remainder of the gland and then finely minced. The mass of minced tissue was next placed within a special press of very powerful design[3] and the juice squeezed from it. The juice was then strained and injected subcutaneously.

Injections were usually made daily for several days. One per cent peptone solution was injected into the control animals.

Fully matured guinea pigs, well housed and nourished, were used as test animals and controls. Copulation was checked by the presence of fresh sperm in the vagina. If mated without the finding of sperm, the females were isolated from the males. Sperm are quickly

Fig. 1. Internal genital organs of immature albino rat, used as control.

[2] Kindly furnished by Parke, Davis & Company.

[3] Manufactured by V. Mueller & Co. Chicago.

[1] Contribution No. 8, Series B, from the Department of Anatomy of the Medical Department of the University of Georgia, Augusta. This work has been aided by a grant from the Committee on Research in Problems of Sex, National Research Council.

destroyed in the vaginal canal and some of the animals became pregnant though sperm were not seen. These were later used for the abortion test. (Unless examination is made fairly soon after copulation, sperm may not be demonstrable.)

It was found that the injection of the peptone solution did not effectively prevent conception, as two of three animals conceived after having received it (see Table I). Neither did the solution produce abortion (see Table II.)

Four series of test animals were used. Two series received the fresh juice of the bovine hypophyses and two the urinc extract. Animals that had just copulated and others of various stages of pregnancy were used.

One series of 11 animals injected after copulation to test prevention of conception received the juice of from three to ten hypophyses. Eight of these animals conceived and 3 died. Since conception was not prevented in any of the animals that survived, no interference with this function occurred. The cause of death in the 3 animals that died was presumably protein intoxication from the injected material (see Table III).

Another series consisting of 4 definitely pregnant animals received each the juice of from three to twelve glands. All of these animals died, probably from the same cause as those listed in Table III. All aborted within a day following the last injection and died within 2 days after that injection. The one hundred per cent mortality in this series contrasts with that of 27 per cent in the preceding series and was probably due to the fact that the animals were pregnant when injected (see Table IV).

TABLE I.—CONTROL ANIMALS INJECTED WITH 1 PER CENT PEPTONE SOLUTION BEGINNING DAY OF COPULATION

Animal No.	Copulation date	Peptone c.cm.	Injections	Total c.cm.	Result
1149	6–5–29	0.5	6	3	Conceived
1175	6–5–29	0.5	6	3	Conceived
1057	6–5–29	0.5	6	3	Etrus 6–23–29

TABLE II.—PREGNANT CONTROL ANIMALS INJECTED WITH 1 PER CENT PEPTONE SOLUTION

Animal No.	Copulation date	Pregnant days	Peptone c.cm.	Injections	Total c.cm.	Pregnancy
1152	2–22–29	28	2½	9	22½	Unaffected
1154	2–28–29	40	5	9	45	Unaffected
1040	3–22–29	42	1	11	11	Unaffected
1127	3–28–29	35	1	12	12	Unaffected
1139	4–3–29	28	1	12	12	Unaffected

A series of 8 females was injected just after copulation (that is, beginning the same day) with the urine extract. Each animal received a total of from 5 to 10 cubic centimeters of the extract, each cubic centimeter containing 50 rat units of hormone, or a total of from 250 to 500 units. Six of these animals conceived and 2 did not. It is interesting to note that there were no deaths in this series. Since all matings cannot result in conception, it can be seen that with a 75 per cent conception rate,

Fig. 2. Genital organs of a mate of the control animal of Figure 1, after injection of 2 cubic centimeters of urine extract daily for 5 days (500 units). Hypertrophy marked.

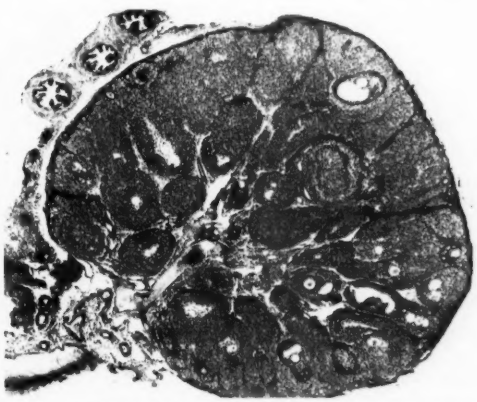

Fig. 3. Photomicrograph of ovary of immature guinea pig after 5 daily injections of 100 units each of urine extract, showing massive luteinization and included ova.

TABLE III. — TEST ANIMALS INJECTED WITH FRESHLY EXPRESSED JUICE OF ANTERIOR LOBE OF BOVINE HYPOPHYSIS JUST AFTER COPULATION

Animal No.	Injections	Each (glands)	Total (glands)	Prevent conception	Died
1501	2	3;2½	5½	No	No
1515	2	1;6	7	No	No
1520	2	2;2½	4½	No	No
1524	1	5	5	No	No
1526	2	3;6	9	—	Yes
1527	2	2;3	5	No	No
1528	2	3;3	6	—	Yes
1529	2	3;3	6	No	No
1530	1	3	3	No	No
1540	1	4	4	—	Yes
1541	2	2;4	6	No	No

TABLE IV.—PREGNANT TEST ANIMALS INJECTED WITH FRESHLY EXPRESSED JUICE OF ANTERIOR LOBE OF BOVINE HYPOPHYSIS

Animal No.	Days pregnant	Injections	Each (glands)	Total (glands)	Aborted	Died
1516	27	2	6;6	12	12–2–30	12–3–30
1519	52	1	3	3	1–3–31	1–4–31
1515	47	1	3	3	1–4–31	1–5–31
1524	41	2	3;3	6	1–4–31	1–5–31

no particular interference with conception was to be observed here (see Table V).

In the last series 14 pregnant animals received each from 5 to 27 cubic centimeters of the extract. Eleven of these animals aborted, 1 delivered at term, and 2 died (see Table VI).

Throughout this work it was found that the urine extract was not stable, and new material was frequently received for carrying on the investigation. The use of deteriorated extract no doubt explains the failure to produce abortion in animal No. 1530 in Table VI, though material was injected that should have contained 1350 units, much more than was necessary in any other animal. Even up to the present the writer does not know of a thoroughly stable preparation of this kind.

To determine the potency of the two products used in making the injections, some of each was injected into both immature guinea pigs and white rats. It was found that the urine extract had the more pronounced effect

TABLE V.—TEST ANIMALS INJECTED JUST AFTER COPULATION WITH EXTRACT OF URINE FROM PREGNANT WOMEN (50 RAT UNITS PER C.CM.)

Animal No.	Injections	Total units	Prevent conception	Died
1501	5	500	Yes	No
1533	5	500	No	No
1536	5	250	No	No
1550	5	500	No	No
1555	5	500	No	No
1556	5	500	No	No
1564	5	500	No	No
1565	3	450	Yes	No

TABLE VI.—PREGNANT TEST ANIMALS INJECTED WITH EXTRACT OF URINE FROM PREGNANT WOMEN (50 RAT UNITS PER C.CM.)

Animal No.	Days pregnant	Injections	Total units	Aborted	Died
1501	44	4	400	Yes	No
1506	56	4	400	Yes	No
1517	44	5	250	No	Yes
1520	44	3	300	Yes	No
1523	39	6	600	Yes	No
1527	52	6	500	Yes	No
1527*	47	6	600	Yes	Yes
1529	61	1	100	Yes	No
1530	41	9	1350	No	No
1536	43	6	600	Yes	No
1541	43	7	650	Yes	No
1550	32	6	600	Yes	No
1556	13	6	600	Yes	No
1557	23	6	600	Yes	No

*Used twice.

in producing hypertrophy of the internal genitals, but that the fresh juice of hypophysis was also active could be seen by its luteinizing effect on the ovaries of the immature animals. The result of the injections of the urine extract is shown in Figures 1 and 2.

Serial sections were made of the internal organs of generation of these immature animals and also of the internal organs of a test animal (No. 1527) that had died after aborting from the injections of the urine extract. In all cases the ovaries showed a luteinization of all follicles to form a compact lobulated mass

Near the centers of many follicles ova could be seen, surrounded by lutein masses (Fig. 3).

The results of these experiments are consistent as far as the effects of the two different materials used are concerned and the interpretation of both results is offered as follows.

As soon as the injections are made, it is probable that stimulation of the follicular apparatus begins, to be followed by the luteinization process. Since about 4 days are required for the fertilized ovum to pass down the tube and arrive in the uterus, it may be that the formation of lutein cells has advanced far enough to elaborate sufficient progestin (Corner and Allen) to take care of the embedding and subsequent nourishment of the early ovum by the time it reaches there. If this is the case, it accounts for the failure of the injections to prevent conception.

The termination of pregnancy may be attributed to the production of estrin by follicle stimulation, for as shown by the work of Parkes and Bellerby, Margaret Smith, and the present author, injections of estrin in sufficient amounts brings normal pregnancy to an end. It is true that Doisy and his co-workers (8) have shown that a highly purified preparation of estrin had no detectable effect on the length of the gestation period of albino rats, but this needs to be confirmed for the guinea pig. It should be remembered that Margaret Smith was unable to terminate gestation in the albino rat after the fifth day with as high as 80 rat units. The fact is that pregnancy was terminated in the present experiments by the injection of preparations that produced a marked terminal luteinization of the ovaries.

If the observation of Doisy and his coworkers just mentioned (that pure estrin does not terminate pregnancy) is true, it must be considered possible that some other element in the injected material may have been the causative agent in terminating the pregnancies. So in this instance the abortions may have been due to some extraneous substance in the extracts.

It is worthy of mention that in terminating pregnancies in the guinea pig with estrin that there was a very high mortality of animals in the second half of gestation, whereas in the present experiments, the mortality after abortion from the urine extract at all stages of pregnancy was quite low. The inference is that the hypophyseal hormone did not upset the balance between the estrin and corpus luteum necessary for the dilatation of the pelvic ligaments of the guinea pig (Hisaw), so that the mothers did not die from sapræmia on account of retained dead fetuses, as suggested by the author (6) in reporting the former work.

SUMMARY

Experimental findings show that injections of freshly expressed juice of the anterior lobe of bovine hypophysis or of extract of hypophyseal-like hormones from the urine of pregnant women into sexually mature female guinea pigs do not prevent conception when administered in serial daily doses beginning the day of copulation, but do cause abortion when given in a like manner to animals in any stage of pregnancy.

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CLINICAL SURGERY

FROM THE LEYSIN INSTITUTE OF HELIOTHERAPY

HELIOTHERAPY AND ORTHOPEDICS IN SURGICAL TUBERCULOSIS

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THE systematic application of sunlight in the treatment of tuberculosis of bones and joints has enabled us to bring about a rational procedure with the object not only to treat the lesion but also to build up the general health of the patient. Although heliotherapy, carefully applied according to the rules of precise dosage and of careful technique, may overcome all local effects of surgical tuberculosis, it does not in itself correct deformities or overcome bad posture. We have found it wise to abandon the use of operations that maim and thus enfeeble the patient's resistance, that may aid the distribution of Koch's bacillus, and that often open the door to serious secondary infection. We have been led definitely to abandon the use of the large plaster apparatus, whether rigid or not, as we believe that its use is a great mistake from the physiological and orthopedic standpoint. Such apparatus keep the affected parts from contact with the air and sun, soften and produce atrophy of the skin as well as of the underlying muscles and bones; with the result that decalcification caused by want of light is added to that produced by the tuberculosis of the bones.

We have replaced the closed plaster cast with a very simple arrangement for fixation and extension, the aim of which is to provide for the affected joints a sufficient degree of immobilization and at the same time to expose them freely to air and sunshine. We have worked out for each part affected a suitable orthopedic arrangement.

As immobilization in the lying position is necessary in the majority of cases, we shall say first a few words in regard to the bed that we use, for the bed constitutes, so to speak, the touchstone of the treatment. Its construction is very simple; the width is 75 centimeters, while the length varies; it is furnished with large wheels mounted on ball bearings which allow the nurse to move the patient without exertion or jarring. The spring mattress is formed of strips of metal with wide spaces to ensure permanent airing of the mattress. The mattress is always flat, very hard, and of uniform consistence. It has the advantage of remaining always dry; a soft mattress more or less wrapped round the lower parts of the body prevents the evaporation of perspiration and thus leads to softening of the skin and the formation of bed sores. To protect the head of the patient during sun treatment a shade made of linen, supported by a movable stem, can be fixed to each bed and its position changed as desired. The construction of the beds has also been modified to meet the requirements for the treatment of the part affected.

POTT'S DISEASE

In all cases in which the vertebral column is affected we prescribe rest in the lying position. Patients affected with Pott's disease, who have no deformity and have good muscular development, simply lie upon the hard mattresses without pillows. But if the patient—and this is the most frequent type of case—reaches us in a state of advanced emaciation and with atrophied muscles, it is absolutely necessary to maintain the proper curvature of the spine, and pillows filled with millet are used; another pillow supports the scapulæ and the spine. When Pott's disease is associated with angular curvature, we try to bring about reduction by applying slow and progressive pressure which is exerted purely by the weight of the body. A pillow stuffed with millet is placed under the kyphosis; later one of sand, and the thickness is gradually increased. At first the pillow should be of somewhat yielding consistence so that the curvature is supported over its whole surface. In this way the formation of scoliosis, which would result from lateral pressure exerted upon the curve, is avoided. When, however, the curvature becomes distinctly less and the skin becomes less sensitive, we replace the sand pillow with a rectangular block of wood, the thickness of which is adapted to the part of the body under which it is to be placed and to the size of the curvature. This has an advantage over the

sand pillow in that it retains its shape and remains completely smooth and dry, two indispensable points in avoiding bed sores which are apt to be caused by moisture and creases. Patients become accustomed quickly to this block and find in it more comfort than in the pillow. When pains have completely disappeared, and from the time that radiography shows the development of vertebral consolidation, we encourage the patients to assume the ventral position. This is the best physiological position, for, by means of exposure to the sun, it provides for restoration of the muscles of the back and thus furnishes the patient with a veritable muscular corset which is the ideal and rational support for the vertebral column. The three pillows used in the dorsal position are now replaced by a single very hard triangular pillow which is slipped under the thorax in order to accentuate the lordosis of the dorsal spine and thus to aid in correcting the dorsal and lumbar curvatures. In cases seen early, this position helps to prevent subsequent deformities of the spine. The patient lying upon the stomach has a tendency to throw back the head in order to look in front of and around him, and he thus increases the physiological lordosis. The movements of the body produced in this way are admirable exercises, as they bring into play the muscles of the scapula, humerus, and dorsal regions—movements which are in no way dangerous, since the vertebræ are not burdened with the weight of the body. The ventral position, in fact, contributes, by the pressure exerted upon the abdomen, toward regulating the functions of the bowels. The patients also prefer this position; they become used to it after only a few days of training and in it spend the greater part of the day and sometimes even of the night. Lying upon the stomach they can undertake light manual work, such as writing, drawing, wood carving, thread work, and typewriting. In Pott's disease with sinuses, the wounds and sinuses, exposed in this way freely to the action of the sun, dry up much more quickly than under any other method of treatment.

In Pott's disease of the cervical spine we use an extension arrangement which exerts pressure only upon the occipital region: a molded celluloid headpiece rests upon a wheeled carrier moved by an extension weight; thus all the region of the chin is free from pressure. The neck region also is freely exposed to the air; any cold abscess or sinus is controlled very early, and the sun has free access to it. This treatment again builds up all the muscles of the affected region; patients recover with complete mobility of cervical spine.

The roentgenograms in cases of Pott's disease treated by heliotherapy are very striking. By them we can follow the course of repair as the vertebræ become progressively hardened, the vertebral blocks becoming solid and often supported by the calcification of a peri-focal abscess, the calcification acting like the nuts which bolt together two pieces of metal.

From the clinical standpoint, the progress of the cure is also interesting to witness. In spite of the fact that the greater number of patients with spinal tuberculosis come to us affected with cervical, dorsal, or lumbar vertebral disease, the process often affecting several parts of the spine, and in spite of the fact that the patients almost always are wasted by having worn plaster apparatus and present angular curvatures. large or small, within 1½ or 2 years—seldom more—they are completely transformed. Their bodies have become uniformly developed, their muscles are powerful, their deformities are corrected, at least partially, by means of a compensatory lordosis above and below the curvature, the residue of the latter being partly hidden by the splendid muscles. It is easy to understand that such robust patients are thus prepared by the work cure to take up normal activities once again.

We have never used the bone graft, for we believe that this procedure prevents or hinders the formation of the vertebral block which is the natural process of cure that is so obviously helped by heliotherapy.

HIP JOINT DISEASE

For disease of the hip, we have always been in favor of continuous extension which is carried out by means of strips of plaster which are fixed along the sides of the thigh, so as to avoid compression of the knee and so as to leave the greater part of the skin uncovered. By means of heliotherapy the muscles are always preserved; they often develop to such an extent that it is difficult to distinguish between those of the affected and the healthy limb. The groove on wheels, which we employ to support the affected limb, keeps the limb in good position and maintains effective extension by means of small weights. The patient is kept in the rational position, that is, the central part of the body is raised by means of a pillow stuffed with millet, so that the pelvis becomes the highest point of the body. This procedure permits easy correction of faulty positions and helps to prevent their formation. Moreover, bed sores and their later serious consequences are prevented. A further advantage is that we are able to control, by means of the traction exerted,

the immobilization of the patient in a manner that is at once necessary and sufficient, for the immobilization becomes less rigid in proportion as the inflammatory symptoms grow less and disappear.

When faulty position of the hip is associated with a tendency to pointing of the foot, we use a special sandal which is furnished with an elastic band along the tibia; this band, stretched more or less according to the amount of pointing, does not in any way prevent movements of the foot, but it does bring the foot back to normal position.

In hip disease especially if there are sinuses situated toward the front of the thigh the lying position sometimes hinders the escape of purulent secretion. Therefore, in order to encourage drainage, we have found it necessary in some former cases in which amyloid degeneration complicated or threatened the progress to get the patients out of bed before cure of the bony lesion was completed, for the drainage of the pus in such a case is of immediate importance. By means of this change of position we have often succeeded in facilitating the evacuation of the pus and as a result, intoxication has been arrested, the temperature lowered, and both the general and local condition of the patient correspondingly improved.

In the majority of our patients with tuberculous disease of the hip, the roentgenograms taken on the arrival at the hospital show an osteo-arthritis in full activity and the acetabulum, the head, and even the neck of the femur show in general a definite disintegration which is represented in the photograph by a soft shadow which completely blurs out the contours of the joint. In the midst of this chaos in time a new head makes its appearance. Its contours, as well as those of the acetabulum, become more definite while the parts affected by atrophy become the seat of an intensive recalcification. In certain cases the head of the femur, partly destroyed, has broken through the caseous edge of the acetabulum, and we can see the progressive reformation of a new joint cavity; the opening is obliterated, the head of the femur is reformed to such an extent that the trabecular structure is outlined with large meshes—an obvious sign of intensive calcification. In this way new joints are formed between the acetabulum and the new head of the femur, thus producing a degree of functional adaptation which could hardly have been hoped for. The frequency of this occurrence (in 85 per cent of cases) is in fact one of the special features obtained by heliotherapy in the treatment of tuberculosis of the hip. This adaptation is brought about not only by the reconstruction of the joint but also by the restoration of the muscles in the thigh; the muscles commence again to play their part as mechanical structures, indispensable to the function of the joint.

The prognosis in hip disease is equally as favorable as it is in Pott's disease; the results are as good in the adult as in the child, and even when the osteo-arthritis is closed, the presence of sequestra in the acetabulum—a not infrequent occurrence—is not necessarily a complication, for the sequestra almost always become reorganized and do not in any way obstruct the function of the joint. It is very interesting to watch in the roentgenograms how the reorganization of these sequestra is effected by the intermediation of small trabeculæ which reunite the larger one in the cavity of the acetabulum. When secondary infection is present the spontaneous elimination of necrosed sequestra often occurs as it does in osteomyelitis. We resort to sequestrectomy only in the presence of sequestra *en grelot* and this procedure carried out always during the period of repair is not as some have stated an "enticement to recurrence." Experience convinces us to the contrary. When there is not only restoration of function of a joint but a good clinical recovery especially when the roentgenograms show recovery, even though this is only partial, the circulation, and therefore the local power of defense, is aided much more than if ankylosis had been brought about, for ankylosis by inactivity encourages the atrophy of bone and muscle. Recurrence more often follows in hip joints which have been ankylosed for some time than in hips which have been kept movable by heliotherapy, provided the patient has quitted Leysin only after his radiographic *exeat*.

TUBERCULOSIS OF THE KNEE

In the treatment of osteo-arthritis of the knee, we combine heliotherapy with extension of the leg. This procedure has the advantage of avoiding compression and ulceration on the articular surfaces and, by supporting the leg, helps to a large extent in bringing about a return of functional movement. In all cases the leg is placed upon an inclined plane to increase the return circulation. We attach great importance to this rational arrangement.

In cases complicated by dislocation of the tibia, we use in addition to extension, suspension of the leg by means of elastic bands from a hoop placed like a bridge above the leg. The weight of the leg helps to correct the faulty position, so that one is never obliged to resort to forcible reduction. If

there is also present a lateral displacement we add traction in the direction opposite to the displacement by means of cords attached to weights. The joint remains always uncovered and thoroughly exposed to the sun's rays.

When the osteo-arthritis, almost always painful, is complicated by infiltration around the joint, by abscesses or by sinuses, giving the joint the appearance of a white spindle, heliotherapy is of special value because it acts in many ways to correct the condition. Heliotherapy at once possesses determinant, soothing, bactericidal, resolvent, and hardening powers. Pain abates and disappears; the parts which are stretched and hard, and shine with smooth atrophied skin, become soft and pigmented; diffuse infiltration lessens and disappears; the outline of the knee cap shows itself anew; and the muscles, which up to now have been flabby and atrophied, resume their true shape. The roentgenograms which are taken at the beginning bear witness to the seriousness of the condition of these bone and joint conditions in which the tissues are dissolving into caseous and purulent material; they show the ulceration of the articular surfaces often associated with secondary infection. In some cases the caries of the bone has given place to the formation of large sequestra; all show the classic thickening of capsular infiltration or fungus degeneration.

Progressive roentgenographic examination of the bone and joint make clear the reconstructive action brought about by heliotherapy. The films show the demarcation and progressive hardening of the foci, the reappearance of bony structure with thick trabeculæ in the blur of caseous disintegration, the reorganization of the sequestra, and the reformation of the joint surfaces, which are demonstrated by the presence of a new joint space. In the same proportion as bony regeneration takes place, the thickening of the capsule abates and disappears, in fact in some cases recovery takes place to such an extent that without exaggeration the joint may be said to be *restitutio ad integrum*. It is easy to understand that in such conditions the functional recovery of the joint is not at all extraordinary. Just as in the case of hip disease, this does not produce any risk of return when the radiographic recovery has been attained. Functional recovery is always spontaneous and we never seek to contribute to it by untimely movements, either active or passive, for such movements are always prone to produce relapses.

Nature herself acts with such discernment that when she judges it right to produce ankylosis (and this is the exception) we take care not to interfere, and we bow to her decision. Age has no unfavorable influence upon the return of function in the joint, for we have seen restoration of function in patients of 60 and even 70 years (thesis of Dr. Miéville). Some roentgenograms which show foci in the form of cavities which are near the epiphyses and are of considerable size and which may affect either the femur or the tibia, show after demarcation the reappearance in the foci of new bony structure which fills up the cavities and gives to the bone an apparently normal aspect. When sequestra lie in these cavities they become organized without difficulty and help to check the process. We have never been forced to operate in such cases, although some such patients had already been operated upon without success. The possible saving of time does not compensate for the risks involved in operation and the danger of secondary infection, which is very great in operations in the immediate neighborhood of the knee joint. A fact which deserves to be mentioned is that when heliotherapy is used in treatment of tuberculosis of the knee it often preserves the movement of the joint, but it may also bring about the desired ankylosis when an excision has unfortunately been followed by pseudo-arthrosis. In support of this statement we may mention a number of cases in which the patients were admitted to Leysin after excision and who were affected by secondary infection and numerous sinuses; 16 in 1 case; 13 in another; and 11 in a third. These patients had pseudo-arthrosis with flail knees, but all recovered with ankylosis after all the sinuses had dried up and cicatrized.

TUBERCULOSIS OF THE FOOT

Of all the joints, the tibiotarsal is the one which gives the most favorable results both from the point of view of curability and from that of restoration of function. In only one other joint are the results more favorable and that is the wrist.

Immobilization in a groove, which leaves the leg and the joint in constant contact with the air and light, and elevation upon an inclined plane constitute, as they do for the knee, the chosen method. Pointing of the toes is corrected or prevented by a sandal joint to the groove. Our apparatus, by mild and progressive traction which is easily regulated, prevents atrophy of muscles and favors the formation of the new joint and the return of joint function. Even when the osteo-arthritis is complicated by secondary infection, and even when there is extensive destruction of bony tissue, recovery in good position and restora-

tion of function, more or less complete, is the rule. We have never had to excise or remove the astragalus—procedures which we regard as a last resort and which are always avoidable if helio-Alpine treatment is used.

Moreover, we have treated numerous cases of disease in the tibiotarsus and metatarsus, of such great extent that amputation of the foot had been considered inevitable, but the patient made such perfect recovery that the affected foot could hardly be distinguished from the healthy one. Radiographic films showed that the joint surfaces which had previously been ulcerated or completely destroyed by the caries were healed and sound.

ARTHRITIS OF THE SHOULDER

In arthritis of the shoulder, we seek to fix the arm in slowly progressive abduction, by means of a hinged apparatus with notches which allows the gradual separation of the arm from the side, and upon which the extension is fixed. This arrangement, which allows free access of the sunlight to the joint, prevents bad position of the arm in relation to the scapula and the loss of compensatory movement of the whole shoulder. It is a well known fact that in this joint more than in any other weakness corresponds to muscular inefficiency, and that osteo-arthritis of the scapula is associated with atrophy of the shoulder muscles. In this as in all joints, heliotherapy not only checks the development of the most extensive destructive processes in the head of the humerus and in the glenoid cavity and completely remedies the defects, but it is also an admirable restorative to the scapulohumeral muscles and greatly helps the return of functional movement. We have never had to resort to excision. The duration of treatment is about 14 months (12 in simple cases of tuberculosis; 16 in osteo-arthritis complicated by secondary infection).

TUBERCULOSIS OF THE ELBOW

For the elbow joint, we use a double groove or a double jointed splint which can be fixed as desired. We endeavor above everything else to bring back the joint to a right angle and maintain it there. Under the action of sun treatment there takes place complete repair of the foci of osteo-arthritis, regeneration of cartilages more or less destroyed, absorption of infiltrations around the joint, and the disappearance of fibrous adhesions. The restoration of the muscles, which is usual, is a great help here as elsewhere, in the return of function which frequently takes place in the presence of tuberculosis of the elbow even when secondary infection complicates the condition. In a recent study of a series of 31 cases of osteo-arthritis of the elbow observed in our clinics, one of our assistants (Dr. Miéville) found that 24 had suffered from sinuses on their admission; of 22 cases of osteo-arthritis 8 left Leysin cured and with normal function, 9 with reduced function, and 5 with ankylosis; in 12 other cases of suppurating arthritis with sinuses, 6 recovered with complete function, 4 with a range of movement from 30 to 70 degrees, 2 with very limited movement.

TUBERCULOSIS OF THE WRIST

Osteo-arthritis of the wrist is treated by immobilization upon a plane combined, according to the case, with extension by means of strips of plaster fixed to the fingers and provided with elastics that can be stretched as desired and fixed under the apparatus. The apparatus is attached by two leather straps passing one across the hand and the other across the forearm; the hand, the wrist, and the forearm thus remain uncovered.

Heliotherapy of tissues of the wrist gives most favorable results, and recovery is always accompanied by return of function. Our results, and those published by our former assistant (Dr. Hussy), show the preservation of the functional integrity of the hand. These results are essential both for æsthetic and social reasons; it is indeed one of the chief advantages of heliotherapy that complete restoration of a joint is accomplished while the classic excision sacrifices the joint.

TUBERCULOSIS OF THE HAND

Tuberculosis of the metacarpus and phalanges is treated by immobilization upon a small splint combined with extension effected by means of a very simple arrangement. The sun cure in these cases, as in those of spina ventosa, produces equally favorable results. Radiography shows *restitutio ad integrum* in both the anatomical and functional sense is almost always the rule prior to secondary infection. Even when the latter has occurred to complicate the case, new joints form without any functional difficulty. Metacarpal bones or phalanges on the road to caseous breakdown or even partly destroyed by caries recover their structure so completely that when the cure is complete the structure appears much more dense than that of neighboring metacarpals and phalanges.

COLD ABSCESSES AND SINUSES

We cannot conclude this chapter on tuberculous disease of bones and joints without saying a few words regarding our method of treating cold abscesses and sinuses.

The formation of the cold abscess is one of the natural phases in the process of treatment of osteo-articular tuberculosis. We regard the cold abscess as a favorable reservoir of antibodies which contributes in large measure to immunization. From the time that the restoration of the "soil" commences under the action of heliotherapy, the bacilli of Koch lose their virulence in it little by little, and the cold abscess is simply a small laboratory where the immunizing bodies are developed. Because we attribute this therapeutic rôle to it, we deprecate its premature puncture. The contents of the cold abscess are in our opinion pre-eminently "good and laudable pus." Such abscesses are always inoffensive, never produce fever, and one has only to recognize the haste with which the organism seeks to refill an abscess prematurely opened in order to understand the importance attached to this by the *natura medicatrix* in its process of cure. Under the action of heliotherapy and in proportion to the state of improvement of the "soil" the cold abscess is absorbed or calcified. We often see cold abscesses arising from bone in the case of tuberculosis of the lumbar vertebræ, which are so voluminous that they partly fill the pelvis, or abscesses arising from disease of the ribs, which stretch out like wallets along the back, and yet these are absorbed completely during the sun treatment without the curve of temperature showing an elevation by even the tenth of a degree.

In tuberculosis of the knee, for example, the appearance of a cold abscess is always a sign of good omen and indicates the commencement of cure in the course of treatment. Pott's disease is associated as a rule with a large or smaller cold abscess which is absorbed spontaneously or calcified, forming thus around the vertebral fusion a structure resembling a set of supporting bolts. These abscesses are inaccessible to the trocar, and this is perhaps the reason why they are able to develop so favorably. If cold abscesses in the dorsal region are allowed to develop without puncture, why should one attack those of the lumbar region which are accessible? In puncturing cold abscesses not only does one take away their immunizing properties but what is of more importance the door is opened for secondary infection. Infection proceeds to develop as it would in the best culture media, and becomes all the more formidable in that there remains no time for local defensive mechanism to oppose the obstacles which may limit the area of infection.

These are the reasons that have led us to advise against puncture and rather to assume a prudent attitude of expectancy, so long as the development of a cold abscess takes place normally. When its helpful rôle of immunization appears to be ended and when, instead of proceeding toward absorption it seems to be attempting to discharge through the skin, then puncture is urgently called for and should be repeated until all danger is removed. Often, on the arrival of a patient for treatment, perforation of a cold abscess is imminent. If the skin has already become too thin and changed to be punctured and if rupture is inevitable, the affected area should be prepared for spontaneous perforation by the application of antiseptics (compresses of alcohol), and this measure of precaution should be continued until the sinus is closed, for above all it is necessary, if possible, that secondary infection be avoided.

The presence of this formidable complication, we cannot repeat too often, changes altogether the prognosis in surgical tuberculosis. While the progress in a closed case of surgical tuberculosis treated by heliotherapy is favorable, both in children and adults and in any locality, its development may become capricious when a secondary infection complicates the picture. A study and comparison of the temperature charts in these two categories of patients emphasizes this point—the curve in the closed cases of surgical tuberculosis shows a most monotonous regularity, while that in the open case is irregular and broken like the lines of an Alpine panorama. In infection with staphylococci or streptococci there is formed a new microbic flora which soon predominates the clinical picture—the development of the infection is speedily manifested by formidable fluctuations, interminable suppurations, retentions, septicæmic fever, intoxication, and amyloid degeneration. The contrast is striking between the appearance of patients affected by closed tuberculosis and open infection—in the former the patient has the color of health and shows a generally flourishing state, with muscles that are often athletic; while in the latter the patient is pale, often has an earthy complexion, flabby muscles, febrile appearance, and recovery is most difficult to attain.

It is well, however, to recognize that there are different categories of cases; all are not equally serious. A sinus, for example, upon a finger or upon the olecranon may be relatively benign if the focus of disease communicates directly with the opening in the skin and offers no possibility of retention. When, on the other hand, a focus is situated in the vertebral column and the pus makes its way along the psoas muscle to end in a sinus in the upper and inner part of the thigh, it

is easily understood that on this long passage with much burrowing septic retention is easy and absorption by the surrounding tissues will cause serious intoxication.

In tuberculosis of the pelvis, for example of the sacro-iliac joint, heliotherapy usually brings about cure in about a year, provided the tuberculosis is closed, and even when a cold abscess has been present. On the other hand, the pelvis becomes one of the most troublesome sites when spontaneous rupture, or a cut by a scalpel, or an unfortunate curettage has introduced secondary infection into a benign cold abscess. The infection becomes entrenched in the deep and inaccessible windings of the sacro-iliac joint and there follows deep retention and suppuration that cannot be dried up. Surgical intervention, when not urgently indicated, alters the organization of the local defense and provides access to secondary infection. Calot has said that "to open a case of tuberculosis is to open a door by which death will too often enter."

The treatment of secondary infection demands special therapy and technique. First, effective drainage for the fistulous passages should be secured, for the success of treatment and the life or death of the patient will depend upon the possibility of securing discharge from the sinuses. It is of first importance to avoid premature closure of the passages. If insufficient, one can dilate the opening with the help of tents and introduce into it drainage tubes of sufficient length to prevent superficial closure; local heliotherapy is abandoned in favor of general heliotherapy, the doses of which must be cautiously determined depending upon the reaction as indicated by the temperature curve. While it is a serious mistake to incise a cold abscess, wide and early incision of a warm abscess is a wise step to insure sufficient drainage and to eliminate septic retained material.

One should not labor under the impression that antiseptics have great bactericidal action, for antiseptics sometimes exercise an action which is prejudicial to the vitality of the tissues rather than detrimental to the germs. If antiseptics are absorbed along with toxins they may act as a contributing factor in impairing the vitality of the kidneys. We have recourse only to the less harmful, such as gomenol, tripaflamine, diathrene, or thiorubrol, but their action is not sufficient to control secondary infection. In some cases, to help drainage we have employed with success continuous drainage by syphon with weak Dakin's solution or with physiological salt solution. While none of the monovalent or polyvalent vaccines recommended by so many authorities has given us any appreciable results, we have had very interesting experiences with sulphur baths (baths of Schinznach and of Lavey, in Switzerland). Once the cleansing has been effected and regular drainage assured, the sun cure is then able to exert its beneficient action.

Let us say one word finally regarding the influence of heliotherapy upon sequestra. Roentgenograms of bones and joints with closed tuberculosis show in a very precise manner how the sequestra dissolve and become absorbed, or more often become organized and incorporated with the living tissue, so that they cause no trouble in the functional recovery of the joints. In some negatives we can follow the progress of complete elimination of necrosed sequestra (it is characteristic of secondary infection that the sequestra become necrosed as in osteomyelitis).

Nature is able, better than the surgeon, to distinguish between the healthy tissues intended to be preserved and the tissues which are irremediably affected and are destined to be eliminated. Roentgenography shows admirably the stages in this process of demarcation. Thus we regard as useless interference by hasty or premature operations which risk, so to speak, stirring up an ant-heap with a stick, and we maintain an expectant attitude so long as nothing interferes with the spontaneous elimination of a sequestrum. On the contrary, we intervene without hesitation when the sequestrum is *en panne* just as we do when the sequestrum is *en grelot*. After sequestrectomy has been effected the resulting cavity is treated with heliotherapy and open air, which help to bring about cicatrization and at the same time avoid secondary infection.

FROM THE LAHEY CLINIC

A METHOD OF DEALING WITH THE PROXIMAL JEJUNAL LOOP IN POSTERIOR PÓLYA ANASTOMOSIS

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THE following plan of management in posterior Pólya anastomosis of the proximal loop of the jejunum to the gastric stump after partial gastrectomy has proved useful and satisfactory in our hands. Others very probably have employed the same plan, although I have not seen it used or described. While I am not interested in claiming priority concerning this plan, I do wish to describe it because I trust that it may prove as useful to other surgeons as it has in our clinic.

Following partial gastrectomy, connection between the small intestine and the stomach may be re-established by a variety of methods, one of the most popular of which is the end-to-side anastomosis of the jejunum to the open end of the resected stomach, an operation which, in this country, is called the Pólya operation.

Various plans and modifications of this procedure have been practiced. Donald C. Balfour has suggested that a loop of jejunum be brought up over the transverse colon and anastomosed to the cut end of the stomach—the antecolic Pólya anastomosis. This plan has proved very useful, but it has two possible drawbacks. Occasionally, in attempting to carry out this step, I have found the length of the jejunal mesentery so short that when it is brought up at a reasonable level over the transverse colon to reach the cut end of the stomach, it produces such pressure upon the transverse colon that, were distention to occur in that structure, there would be danger of obstruction. It is true, however, that in a large percentage of the cases, the jejunal loop can readily be approximated to the stomach without difficulty, so that this plan has been accepted as very useful. The second possible drawback is, in order that the jejunal loop will reach over the transverse colon, a long jejunal loop must be used, thus resulting in the dumping of gastric contents into a relatively low segment of the jejunum. In all probability this criticism is not serious but, other things being equal, an endeavor should be made to approximate the stomach to the small bowel as near to the duodenum as possible. It must be assumed that the farther from the duodenum the point of anastomosis between the stomach and jejunum be made, the less adapted that segment of bowel will be to receive gastric contents and the greater will be the likelihood of recurrent gastrojejunal ulcer at the suture line.

I have always been inclined to use the posterior Pólya type of anastomosis, passing the loop of jejunum through the transverse mesocolon, thus permitting the anastomosis of the jejunum to the cut end of the stomach at a level somewhat closer to the duodenum. In this manner the occasional difficulty with a short mesentery to the jejunum is avoided and approximation to the cut end of the stomach is made easy. I do not describe the plan here submitted as an argument against the antecolic Pólya plan of anastomosis but rather that it may be available for those who are interested and wish to employ the posterior Pólya type of procedure.

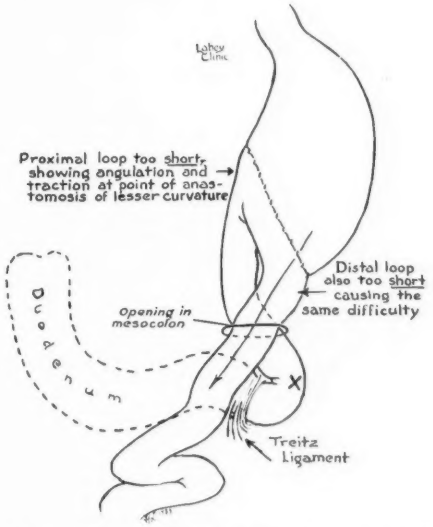
Lahey Clinic
Proximal loop too short, showing angulation and traction at point of anastomosis of lesser curvature
Distal loop also too short causing the same difficulty
Opening in mesocolon
Duodenum
Treitz Ligament

Fig. 1. This diagrammatic figure demonstrates that when an insufficient amount of proximal jejunum is utilized and the stomach is permitted to drop back into the left upper quadrant, traction occurs on the suture line due to the short proximal jejunum. The disadvantage of too short a distal loop of jejunum is likewise illustrated. Note the ligament of Treitz and that, by incision of this ligament, the proximal loop of jejunum may be transplanted above the mesocolon.

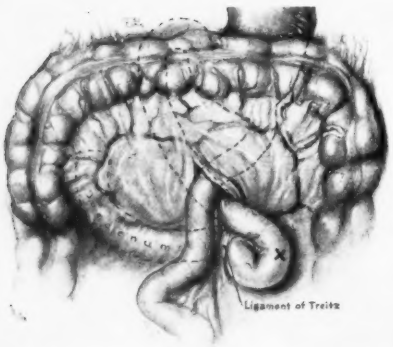

Fig. 2. Drawing showing the angulation of the jejunum if the proximal loop is carried up through the mesocolon and the ligament of Treitz is not severed; also the double barrelled effect of proximal and distal loop of jejunum as they pass through the rent in the mesocolon. Even if the mesocolon is sutured to the gastric stump, as is frequently done, considerable angulation of the proximal jejunum results. Note the ligament of Treitz, which in the plan here described, is to be incised, thus permitting the displacement of this proximal loop of jejunum through the mesocolon so that it runs above it. Then but a single loop of jejunum, the distal one, emerges through the rent in the mesocolon. Note the level of the proximal jejunum marked *x* in order that it may be compared with this point when transplantation has been done above the transverse mesocolon by the plan described in the text.

Fig. 3. This figure is the same as Figure 2, except t at the relations above the mesocolon with the transverse co n turned down are seen. The angulated proximal loop of e- junum is shown below the mesocolon in dotted lines.

Note again the point marked *x* in the proximal jejun n beneath the transverse mesocolon.

In the posterior Pólya type of anastomosis n partial gastrectomy, one of two plans may e employed in dealing with the proximal and dist l loops of jejunum. After the end-to-side anasto-

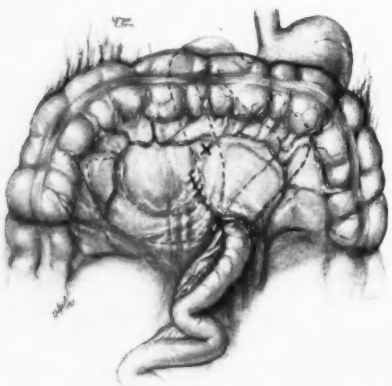

Fig. 4. After incision of the ligament of Treitz and transplantation of the proximal loop of jejunum, the single loop of distal jejunum emerges from the opening in the transverse mesocolon. The mesocolon has been sutured above the single loop of jejunum. The transverse colon is turned up so that the proximal jejunum is shown in dotted lines, indicating its position above the transverse mesocolon. Note now the position of *x* well above the mesocolon and as relates to the level of the jejunum at which the anastomosis has been made.

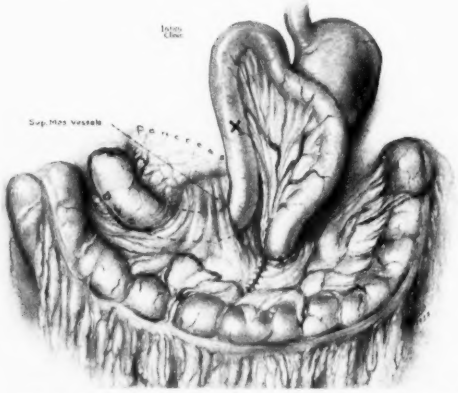

Fig. 5. This figure is the same as Figure 4, except th t the transverse colon has been turned down in order th t the proximal and distal loops of jejunum may be viewe l from above the mesocolon. Note the rent in the mesocol t sutured. One side of this rent represents the incised li - ment of Treitz. Note the point *x* on the bowel now w l above the transverse mesocolon; the lack of angulation a l that more than enough proximal jejunum has been utili l in order to prevent traction and angulation when the sto - ach retracts into the left hypochondrium. Note also th t but a single loop of jejunum, the distal loop, passes throu t the transverse mesocolon.

Fig. 6. Gastro-intestinal tract after partial gastrectomy and Pólya anastomosis by the plan here described. The patient was first given a bismuth enema to demonstrate the position of the colon and then bismuth by mouth to demonstrate the relation of the proximal and distal loops of the anastomosed jejunum to the bismuth filled colon and mesocolon. There are several interesting points in these pictures: The distal loop of jejunum is long and angulates at the point marked *x* where it passes as a single loop through the mesocolon. The proximal loop of jejunum is unfilled with bismuth in both pictures. The caliber of the end-to-side anastomosis between the large transversely cut end of the stomach and the small intestine eventually becomes of quite moderate size.

Fig. 7. Bismuth was given by mouth only, the bismuth enema being omitted. This permits clear visualization of the bismuth filled distal loop of jejunum, and clearer demonstration of the above-mentioned points. Again note part *x*, where the distal loop of jejunum passes through the mesocolon, and the single loop of jejunum passing through the mesocolon and the proximal jejunum unfilled with bismuth.

mosis between the jejunum and stomach, one plan is to suture the slit in the transverse mesocolon anteriorly and posteriorly to the stump of the stomach itself, thus placing the proximal and distal loops of jejunum entirely below the transverse mesocolon in the general peritoneal cavity. A disadvantage of this method is that if the gastric resection be high and the gastric stump short, it will be difficult to bring the transverse mesocolon up high enough so that it can be sutured to the gastric stump without distortion of the transverse colon. In some very high gastric resections, in our hands, it has proved impossible.

The other plan customarily employed is to bring both loops of jejunum up through the transverse mesocolon, to permit the stomach to retract to its natural height, and then to suture the rent in the transverse mesocolon about the double barrelled loop of proximal and distal jejunum (Fig. 2). The disadvantages of this procedure are that the jejunum must emerge from its retroperitoneal position in the jejunal fossa, the afferent loop must ascend through the slit in the transverse mesocolon, and the efferent loop must also emerge through the transverse mesocolon, thus producing a double barrelled effect with considerable angulation of the loop of proximal jejunum. Another undesirable factor in the plan is that it is not possible satisfactorily and accurately to close the opening in the transverse mesocolon when two loops of jejunum pass through it.

By the plan we have employed and here suggest, an opening is made in the transverse mesocolon usually just to the left of the root of the ligament of Treitz. In some measure the site of the opening in the mesocolon must depend upon the location of the middle colic artery. Through this aperture there is passed a loop of jejunum which is sufficiently long so that when the stump of the stomach retracts upward into the left hypochondrium, there will be no undue traction and tension upon the point where the proximal loop of jejunum is attached to the cut end of the stomach at the point which marks the lesser curvature. I

wish particularly to warn operators who are not familiar with this step that, in order to avoid tension as here described, the proximal loop of jejunum must always be a little longer than at first seemed necessary. This is extremely important, since, if the end-to-side anastomosis has already been made and following release of traction upon the stomach and the ascent of that organ, there is tension on the upper angle of the suture line due to too short a proximal jejunal loop, a dangerous and distressing situation arises (Fig. 1).

Having satisfactorily completed the posterior Pólya anastomosis, the plan which I have employed and wish to describe is as follows:

The ligament of Treitz is cut from its lowest insertion into the jejunum, up to its origin in the mesenteric root. This permits of mobilization of the uppermost part of the jejunum, so that the proximal loop of jejunum now anastomosed to the stomach can be passed up through the slit made in the transverse mesocolon, and in this way the entire proximal loop of jejunum is brought above the mesocolon and is excluded from the greater general peritoneal cavity. While the true vascular root of the transverse colon is still above the junction of the jejunum with the duodenum, nevertheless there is less angulation of the proximal jejunum than when the proximal jejunal loop enters the greater peritoneal cavity at the jejunal fossa and is again passed upward out of the greater peritoneal cavity through a slit in the transverse mesocolon. It has the additional advantage that now but a single segment of bowel, the distal jejunal loop, emerges through the transverse mesocolon (Figs. 4 and 5). Snug suture of the slit in the transverse mesocolon about the single loop of bowel is thus possible and the danger of hernia through this slit is lessened.

The plan of the placing of the entire proximal loop of jejunum above the transverse mesocolon in a posterior Pólya anastomosis has, if anything, not added to, but diminished, certain of the technical difficulties of this type of anastomosis after partial gastrectomy. In all of the cases in which it has been employed, the anastomosis has functioned well and any question of the need of entero-enterostomy between the proximal and distal loops of jejunum has been eliminated.

The plan submitted has proved valuable by actual repeated employment. It is not suggested that it be employed in preference to other methods but it is hoped that it may prove useful to those who like and employ the posterior Pólya type of anastomosis after partial gastrectomy.

RENAL TUBERCULOSIS

Diagnosis and Treatment, with Study of Ninety-Seven Cases of Nephrectomy for Tuberculosis[1]

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THE frequency with which tuberculosis of the kidney occurs compels the general practitioner as well as the urologist to be constantly aware of its possible existence. It is estimated that 0.5 per cent of all surgical operations and 27 to 30 per cent of kidney operations are performed for renal tuberculosis. Great importance is therefore attached to renal tuberculosis in the field of urological surgery.

It has been said that, by careful study and investigation, an undiagnosed case of renal tuberculosis can be ferreted out in almost any large clinic. This is true because the subjective symptoms may be absent or so variable as to make the presence of tuberculosis unsuspected. At the present time, however, the diagnosis is more frequently made, partly because we are constantly searching for this disease but primarily because of the recent advances in the art of urological diagnosis.

It is not my intention to dwell on the many disputed points in etiology, pathology, diagnosis, and treatment; these subjects have been so thoroughly discussed that a repetition would be superfluous. Moreover, it seems to me that most of the debatable questions have been sufficiently solved to give us a firm foundation upon which to work. Much experimental and clinical evidence, by careful observers, has clarified many erroneous conceptions of renal tuberculosis. I do wish, however, to emphasize a few points in the diagnosis and treatment, most of which have been mentioned but which will, I feel, stand repetition. These points are based mostly upon a study of 97 cases upon whom nephrectomy was performed for tuberculosis of the kidney in the New York Hospital, between December, 1914, and July, 1932.

Until further convincing proof to the contrary is forthcoming, we feel that some types of tuberculosis of the kidney will heal under certain circumstances. It is also our impression that "tuberculous bacilluria" without renal pathology does not exist, and that when such cases are reported the error lies with the pathologist. We believe, too, that removal of a kidney from which tubercle bacilli have been repeatedly recovered, *without* other clinical evidence, is an error in judgment; that genito-urinary tuberculosis almost invariably enters the kidney by the blood stream and secondarily affects the ureter, bladder, and genital tract; that bilateral renal tuberculosis occurs more often than was formerly believed, and that in some patients, in whom bilateral renal tuberculosis has been diagnosed, removal of the destroyed kidney offers the greatest hope of benefit to the patient.

We follow the general principles mentioned chiefly because of the excellent experimental work reported by Medlar, Harris, Uffreduzzi (quoted by Forni, 10), Helmholz (quoted by Braasch, 4), and others. These facts have also been fairly well established by many thorough clinical observers, among whom may be mentioned Thomas (26), Braasch (3), Beer, Stevens, Kretschmer, Bumpus, and Wildholz. Together with our own observations, we feel justified in assuming these general principles as a working basis, subject to any unusual findings in a given case.

PRECLINICAL TUBERCULOSIS OF THE KIDNEY

Let us first consider the preclinical, or silent stage, of renal tuberculosis. Harris, in his excellent experimental work, ably describes this stage by saying: "Of the course of the disease from the onset of symptoms to its termination by operation or death, one is thoroughly familiar; but of the course of the disease from the time the first tubercle bacillus is implanted in the kidney, until the involvement of the bladder gives rise to symptoms which attract attention to the urinary tract, nothing is known." He studied the urine of 43 adults and 67 children who had bone tuberculosis and obtained tubercle bacilli in 37 per cent of the adults and about 13 per cent of the children. Of these, 68 per cent exhibited none of the usual symptoms of renal tuberculosis. Furthermore, several patients, whose urine for a year or more showed tubercle bacilli and pus, are now free from symptoms and show no tubercle bacilli in the urine. He believes that this silent stage usually lasts for years. Herman (12) expresses the eventual outcome of some of these patients when he suggests that "unilateral surgical (tuberculous) lesions represent the progressive remains of a

[1]Read before the Southwestern Branch of the American Urological Association, Denver, Colorado, August 17 to 19, 1932.

Fig. 1. Shows kidney partially destroyed by tuberculosis and with poor function. Note the infected glands just to the right of the fourth lumbar interspace on the left side. This kidney was removed almost 2 years ago. The other kidney (Fig. 2) was also infected with tuberculosis but was functionally perfect. With postoperative medical treatment patient is almost free from symptoms and has gained weight.

once disseminated infection," and, further (13), "In employing the term, primary renal tuberculosis, one refers to a state of primarity of the infection only in respect to the site of its initial appearance in the urogenital system." Hobbs, according to Stevens, found tubercle bacilli in the urine of 8 per cent of 422 patients, with no other symptoms. Bumpus and Thompson also reported 8 per cent of 345 patients who were otherwise symptomless yet had tubercle bacilli in the urine, 23 of whom had had a tuberculous kidney removed, operation in the remaining 5 cases being deemed inadvisable. Thomas (26) states that many cases of renal tuberculosis go undiscovered and heal before destruction occurs. He has also found that no symptoms were present in the majority of cases with early renal tuberculosis.

It is our impression also that this preclinical stage of renal tuberculosis exists much more frequently than has been recognized in the past. We might ask ourselves the difficult question of how

TABLE I.—AGES OF NINETY-SEVEN CASES UNDERGOING OPERATION

Ages in years	Number	Per cent
11 to 20	11	11.4
21 to 30	37	38.0
31 to 40	31	32.0
41 to 50	13	13.4
51 to 60	5	5.2

Youngest patient 12 years, oldest, 60 years, average age, 30.4 years.

TABLE II.—SEX OF NINETY-SEVEN CASES UNDERGOING OPERATION

	Number	Per cent
Total operated upon	97	
Males	50	52
Females	47	48
Patients with postoperative data	73	
Deaths	9	
Males	7	14
Females	2	4

such a condition should be suspected. Barnett states that positive diagnosis in an early case is difficult because the process has not yet punctured the mucous membrane. A. R. Stevens (quoted by Beer) points out that one cannot exclude a tuberculous focus in a kidney because one can prove that such a tuberculous focus exists only after it has broken into the renal pelvis. A careful history and physical examination should be helpful, especially the periodic health examination. Howard long ago stressed the importance of a thorough urine analysis in such cases. It might be helpful to have stained and injected into guinea pigs the urine from individuals who, between the ages of 20 and 40, show white blood cells and a trace of albumin in the routine urine examination. Certainly, as Munger suggests, an individual in early adult life, complaining of polyuria with some dysuria and a little pus or albumin in the urine deserves to be considered a suspect for renal tuberculosis.

I do not wish to suggest that one should become more diligent in the search for renal tuberculosis for the purpose of performing more surgery. Quite the contrary is true, because it would be reasonable to assume that by early diagnosis and proper medical care many kidneys might be saved from probable destruction by progression of the tuberculous disease. Even if this were not possible, certainly by early surgical treatment the prognosis would be much better and the patient saved from the distressing bladder symptoms we so frequently see.

CLINICAL TUBERCULOSIS DATA FOR DIAGNOSIS

The picture of clinical renal tuberculosis may be quite varied. However, in a large percentage

TABLE III.—STATISTICS OF CASES WITH POSTOPERATIVE DATA

Age, years	Number	Dead	Per cent
11 to 20	9	2	22.2
21 to 30	26	4	15.4
31 to 40	26	2	7.7
41 to 50	9	1	11.1
51 to 60	3	0	

of cases it is so nearly the same that the general practitioner should at least anticipate the disease. In the unusual cases it may be quite difficult or even impossible to suspect the underlying cause.

A carefully obtained history in any surgical condition cannot be too frequently stressed. This is particularly true in tuberculosis. Kretschmer has shown that 20 per cent of his 221 cases gave a history of some previous operation for extrarenal tuberculosis. The family history may also be helpful. The development of tuberculosis of the epididymis, either prior or subsequent to the infection of the kidney on the same side, is a frequent occurrence.

Age. More than half of the patients suffering from chronic renal tuberculosis in any series of cases are between the ages of 20 and 40 years; hence it is considered a disease of early adult life. In our series of cases 38 per cent occurred between 21 and 30 years of age, and 32 per cent between 31 and 40 years. Thus, 70 per cent of these operative tuberculous kidneys were in patients between the ages of 20 and 40 years. This percentage coincides with the findings of most observers (Table I).

Sex. In our series there were 50 males and 47 females. Similar data from other authors show that tuberculosis of the kidney shows no marked affinity for either sex, and it is our opinion that it affects male and female about equally. The right kidney was operated upon in 51 patients and the left in 46 (Table II).

Symptoms. Patients with renal tuberculosis seek assistance at a comparatively late stage of the disease. This is true because of the usual insidiousness of the onset and the fact that the primary symptoms are not alarming. Then, too, symptoms referable to the bladder are prone to periods of regression, and the palliative treatment, which usually has been instituted, receives the credit.

In our most recent series of 34 patients, symptoms had been present from 2 months to 7 years, with an average of 20.7 months, before the patients presented themselves for examination. Of these 34, all but 5 complained of some bladder symptoms. Pain referable to the kidney was also a very frequent complaint, occurring in 26. This pain was usually dull or aching in character, and

Fig. 2. Pyelogram of left kidney of same patient as in Figure 1.

sometimes only a feeling of heaviness. It seems worthy of note that pain should have occurred in as many as 76.5 per cent of these patients. In many it was the primary symptom, and in a few the only complaint (Table III).

Intravenous urography. Since the introduction of uroselectan, another distinct aid in diagnosis has been added. This is particularly true in those cases in which it is impossible to catheterize one or both ureters. It is not only helpful in outlining the kidney pelves but also gives us considerable information regarding the function of both kidneys. Unfortunately, however, the kidney pelvic outline is usually not so clear cut and distinct as the outline we obtain by retrograde urography, and it fails entirely to outline the minor deformities in early cases. Intravenous urography has been of diagnostic aid in 12 of the 22 patients in whom it has been used.

Cystoscopic findings. If intravenous urography and the general examination, which includes repeated urine analyses, have not yielded sufficient information concerning both kidneys (which is usual), the patient is subjected to a complete cystoscopic examination. This includes a thorough examination of the bladder, catheterization of both ureters (when possible), with urine specimens

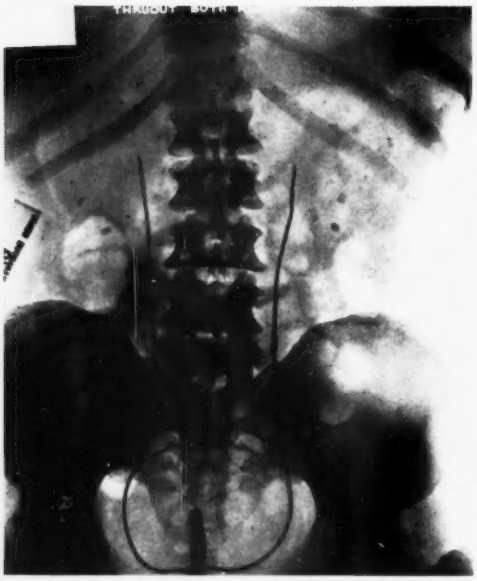

Fig. 3. Plain X-ray picture showing multiple small calcifications in both kidneys caused by tuberculosis. These calcifications are significant of nature's attempt to heal the disease.

collected from the bladder and from each kidney for culture, urea, and microscopic examination, guinea-pig inoculations, and potato culture. One cubic centimeter of phenolsulphonephthalein is then injected intravenously; a note is made of the time of appearance from each kidney, and a determination of the total amount secreted in 10 minutes after the first appearance. Thus, the urea and the phenolsulphonephthalein determine the function, and the guinea-pig and potato culture prove the presence of tubercle bacilli. A plain X-ray picture is taken of the bladder, ureters, and kidneys with the X-ray ureteral catheters in position, followed by a pyelogram of each kidney and a pyelo-ureterogram with the patient in the sitting posture.

DIAGNOSIS

There is considerable difference of opinion as to how much data is required to make a diagnosis and outline the treatment in a given case of renal tuberculosis. Beer states that the cystoscopic picture, together with the finding of tubercle bacilli in the urine, is sometimes sufficient for diagnosis. He also believes that one should obtain positive smears for tubercle bacilli in 89 per cent of cases; that functional tests are significant only late in the disease, and that pyelography is rarely necessary and often harmful. He grants the occasional necessity for exploratory operation. Braasch and La Pena (4) state that diagnosis of renal tuberculosis is more difficult than it was 25 years ago, because in most cases there is less pathology present. They base their diagnosis on evidence of renal destruction as noted by deformity in the pyelogram, but state that the phenolsulphonephthalein functional test, positive guinea-pig, and uroselectan are all helpful at times. Wade calls attention to diminished bladder capacity in 97 per cent of cases. Pyelograms are more important than functional tests according to Kretschmer, who also believes one should not be in haste to operate but should repeat the various tests and pyelograms to prove that the other kidney is free from disease. He states that such delay has never led to a bad result or to any regret. Jeck, in reporting 58 cases from Bellevue Hospital, obtained pyelograms in only 14, 10 of which were typical enough to warrant operation, and expressed fear of pyelography in these cases. Thomas (26) found the pyelogram to be the first positive localizing finding in 56 of 170 pyelograms studied. He always makes bilateral pyelograms and finds a filling defect usually at the tip of a calyx, often so small as to be easily overlooked. Pyelograms were helpful in making a diagnosis in only 26 per cent of Stevens' cases; while Rohrer states that they are unnecessary. Caulk made a diagnosis of tuberculosis in 78 per cent of his cases unaided by pyelograms, but also proceeds with urography, though he states it is not helpful in many cases.

The diagnosis of renal tuberculosis is sometimes exceedingly difficult, and we therefore feel that every possible positive or negative evidence is of the utmost importance. Our aim is not merely to determine that tuberculosis of the urinary tract exists but to ascertain exactly where the infection is and to what extent destruction has occurred. Furthermore, we are anxious to obtain this information as early as possible so that definite treatment may be instituted to relieve the symptoms, as well as to prevent extension and further destruction. It is true that in certain lesions which progress slowly there may be no hurry to operate, but how are we to determine in any specific case how rapidly the process may extend? It is with this in mind that we subject patients to a thorough routine examination and complete cystoscopy.

There is always the danger of carrying tubercle bacilli to the uninfected kidney by passing a ureteral catheter through the infected bladder. Also, a vesicorenal reflux may occur, which would permit one to obtain infected urine from a healthy kidney. Beer believes that these incidents are

Fig. 4. Bilateral pyelogram of Figure 3 showing diffuse tuberculosis of both kidneys. The kidney destruction for such an infection is not great. This patient is at present inoperable and is being treated medically.

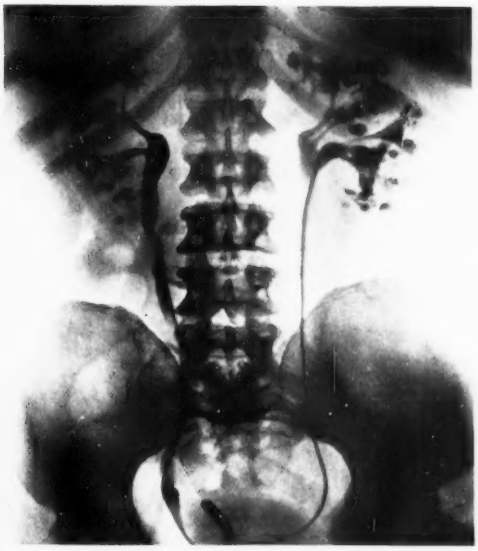

Fig. 5. Bilateral pyelogram of same patient (Fig. 4) taken 6 months after medical treatment. Note that the filling defects are somewhat smaller, possibly due to healing within the kidney. He is free from symptoms most of the time, with occasional remissions of bladder symptoms. His general condition has improved and he is able to continue his employment as a policeman.

common and are occasionally the cause of an erroneous diagnosis of bilateral renal tuberculosis. Thomas (26) has passed catheters to the kidneys of patients with tuberculosis, filled the bladder with methylene blue, lowered the head of the table, asked the patient to strain, and looked for the blue dye to come from the catheters. He obtained the dye in 1.5 per cent of cases. No doubt bacteria may be carried up the ureter with a catheter, or a reflux may occur which permits one to obtain bacteria from a healthy kidney. Still, we feel that the information obtained from cystoscopy and careful catheterization of the ureters far outweighs the possible harm to the patient. Reflux up a *normal* ureter must be rare.

We have come to rely primarily for diagnosis upon the irregularity or filling defect in the pyelogram. Usually other data are necessary for a correct diagnosis, although in the most recent 34 cases the pyelograms were considered diagnostic in 28, or 82.3 per cent, with an error in diagnosis in 2 cases. By means of pyelography, one obtains more evidence of renal destruction than by other diagnostic means. It is true that occasionally a large pyonephrotic sac or a renal neoplasm may simulate tuberculosis, but in these cases removal of the kidney is the only way to ascertain the pathological process present.

There are two common types of advanced renal tuberculosis which may ordinarily be diagnosed by the pyelogram. The first appears as a large, dilated, irregular kidney pelvis and a megaloureter. This type usually has very poor renal function and is the end-result of a continuously destructive process. The second type is characterized by shagginess and irregularity of the major and minor calyces, without much increase in the size of the kidney pelvis, often with calcifications in the kidney substance, and a small, irregular, beaded ureter. This latter group includes those cases which show a tendency toward healing, and usually has a better resistance to the tuberculous infection.

Kidney function tests proved helpful in 26, or 76.5 per cent of our most recent 34 cases. Diminished renal function, as determined by urea, phenolsulphonephthalein, indigo-carmine, or uroselectan, gives us an idea of the approximate destruction in the kidney. In early cases, in which very slight damage has occurred, these tests are often valueless, but by the time the urologist is permitted to examine these patients some kidney tissue has usually been destroyed.

Fig. 6. The filling defect in the left upper calyx is due to tuberculosis. A left heminephrectomy was done, followed by a urinary fistula. One year later a complete left nephrectomy was necessary. This portion of the kidney also showed tuberculosis. At present patient is free from symptoms and has gained weight. (The filling defect in the right pyelogram is due to incomplete injection.)

We rely more upon the percentage of phenolsulphonephthalein excreted in a given time from each kidney than upon the other kidney function tests.

The bladder picture on cystoscopy frequently is sufficiently characteristic to convince one of the presence of tuberculosis. This is very helpful and was present in 25, or 73.5 per cent, of our cases.

Cathelin states that it is impossible to catheterize both ureters in 50 per cent of cases. This may be true at the first examination. In this series, both ureters were eventually catheterized in 28, or 82.3 per cent, although in some cases more than one cystoscopy was required. Occasionally, the catheters could be passed only part way up the ureter.

Finding tubercle bacilli in the urine is very helpful in making a diagnosis, and we search diligently for them by stained smears, guinea-pig inoculations, and Corper's potato culture. They were discovered in 16, or 47 per cent, of the patients. However, we do not hesitate to diagnose renal tuberculosis in the absence of tubercle bacilli in the urine.

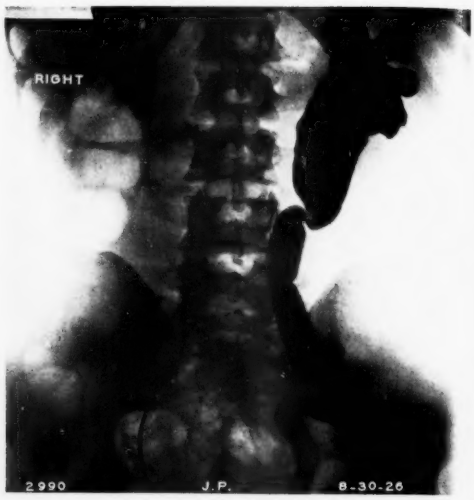

Fig. 7. Shows advanced destruction caused by tuberculosis.

TREATMENT

Renal tuberculosis should be considered a localized infection in an individual who has a general systemic tuberculosis, for it would be unreasonable to assume that a kidney could become infected with tuberculosis without the presence of some other focus of the disease in the body. This fact is particularly important in outlining the treatment of this disease. Although our attention is focused on the renal infection, we should remember that the disease is present in some other tissues even though it may be quiescent at the moment. Hence, it would be reasonable to assume that the patient should be treated in the same manner as one suffering from tuberculosis which had manifested itself elsewhere.

So called preclinical tuberculosis should be treated medically. If no renal destruction has taken place, which can usually be determined by the pyelogram, the patient should have the benefit of careful medical care in the hope that the kidney will heal. If a destructive lesion supervenes during medical treatment, surgical measures should at once be instituted.

Destructive unilateral renal tuberculosis is always surgical, and the offending organ should be removed as soon as the diagnosis is made, unless the patient's general condition contra-indicates surgery. Occasionally, even in the presence of an open pulmonary tuberculosis, or other systemic disease, it may still be advisable to remove the kidney. As Hunt has pointed out, "In the absence

Fig. 8. Early tuberculosis of the right upper calyx. Nephrectomy resulted in complete cure. Note distortion due to granulations, nature's attempt to heal.

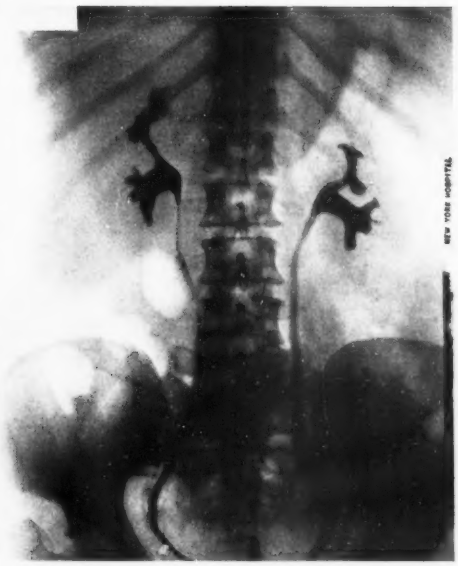

Fig. 9. Early tuberculosis of the right upper calyx. Because of the destruction, a nephrectomy is more urgent in this case than in Figure 8.

of general indications, if the tuberculous kidney is the most significant lesion, its removal is justifiable." Since it is possible to remove a kidney under regional or spinal anæsthesia, the operation assumes much smaller proportions, both as to mortality and morbidity. In rare instances it may be advisable to treat this type of patient medically before operation in the hope that his eventual postoperative convalescence may be shortened. It is our custom to begin medical treatment as soon as the diagnosis is established and continue this treatment during the operative convalescence, as well as after operation.

There is considerable difference of opinion concerning the treatment of bilateral renal tuberculosis. Braasch (3) states, according to Bumpus, that when nephrectomy has been performed, and, following this, urine from the supposedly normal kidney is found to contain tubercle bacilli, the subsequent mortality and the subsequent improvement are practically the same as in those with, as far as can be determined, strictly unilateral involvement. Thomas (26) believes that the removal of one kidney in a bilateral tuberculous infection, is not good surgery. Spitzer admits that some circumstances warrant removal of one kidney, even when the other is known to be affected, but that these occasions are rare. Caulk, Beer, Kretschmer, and others are of the opinion that patients with questionable bilateral renal tuberculosis should not be operated upon until it is satisfactorily established that one kidney is uninfected. They also believe that a diligent search should repeatedly be made for tubercle bacilli from the urine of the normal kidney, and if none are found the case becomes surgical. Folsom strongly advocates the immediate removal of a proved tuberculous kidney, provided a clear urine and a normal function, determined by the excretion of indigocarmine or phenolsulphonephthalein, are obtained from the remaining kidney. McCarthy (quoted by Folsom) believes "it is a much less error to remove any number of tuberculous kidneys, in what subsequently proves to be a bilateral renal tuberculosis, than it is to inoculate even one healthy kidney with this disease by overzealous instrumentation." Uffreduzzi, according to Forni, has found that animals with bilateral renal tuberculosis, which had a unilateral nephrectomy, lived longer than control animals, and that in some cases the progression of the disease in the remaining organ was very gradual. He states that in several instances there was a regression which resulted in a clinical cure, healed lesions being found at autopsy. He explains this as resulting from an increased blood supply to

Fig. 10. A diagnosis of right renal tuberculosis was made and nephrectomy performed. Pathological report showed an infected hydronephrosis but no tuberculosis. This patient's sister showed a similar condition, which was tuberculosis, and helped lead us into this diagnosis.

Fig. 11. Due to this filling defect, three diagnoses were made: (a) Tuberculosis, (b) non-shadow-casting stone, and (c) neoplasm. Only symptom was painless hæmaturia. Nephrectomy revealed a neoplasm surrounding the ureteropelvic junction and extending a short way down the ureter.

the remaining kidney which had a beneficial influence on the tuberculous disease.

Bilateral renal tuberculosis usually manifests one badly destroyed kidney, while the other kidney is only slightly infected. To determine whether or not the second kidney is infected, many surgeons search diligently for tubercle bacilli. In recent years a much larger percentage of bilateral infections has been discovered. After making a diagnosis of bilateral renal tuberculosis, most surgeons relegate the patient to a miserable existence and, as statistics show, to almost certain death in a short time. There have been patients (Fig. 1) suffering from bilateral renal tuberculosis, who have had one kidney removed, and at present the disease is at least arrested and there has been clinical improvement. Similar cases have been reported by Braasch (4), Munger, and others. If even an occasional patient is benefited and his life prolonged and made more endurable, is not removal of a kidney, the infection of which prevents any possibility of convalescence, justifiable? Medical treatment with surgery is very helpful, because, as often pointed out, if relative immunity cannot be built, prognosis is unfavorable.

I do not wish to convey the idea that all patients with bilateral renal tuberculosis should be subjected to operation. There are some patients presenting frankly destructive lesions in both kidneys, and it would be exceedingly difficult to decide which one to remove (Fig. 4). However, I believe that if a destructive lesion is definitely proved in one kidney and the functional test of the other is satisfactory, even though tubercle bacilli have been found in the urine from it, the patient should have the destroyed kidney removed at once. It is our custom to remove the destroyed kidney even when there is pyelographic evidence of a very small infection in the opposite kidney. Inasmuch as the poor outcome in cases in which surgical treatment has been denied has been amply proved, and whereas certain patients are definitely benefited by surgical intervention, we would prefer to make an error of commission rather than one of omission. We have also come to believe that no patient should ever be told that his condition is inoperable. Under proper care the conditions often change, and in two instances patients who were at first pronounced inoperable were later deemed operable; then, because of the earlier in-

TABLE IV.—ANÆSTHETIC USED

Type of anæsthesia*	Number	Average days in hospital after operation	Immediate results			Died
			Arrested	Improved	Unimproved	
Paravertebral	25	18.9	20	3	1	1
Paravertebral and inhalation narcosis	23	22	14	8	1	
Inhalation narcosis	33	29	29	1	2	1
Avertin	2	15.5	0	2	0	
Spinal	14	21.4	6	7		1
All types	97	23.6	69	21	4	3

formation, they both refused operation. They have since died. In these cases there was an apparent regression of the disease in one kidney, while the other became so badly infected that the general health of the patients was impaired. It is difficult to guess what the outcome might have been had they submitted to the operation, but, realizing the hopeless outlook otherwise, heroic measures seem justifiable.

With all the present methods of diagnosis, we feel that an exploratory diagnostic operation should rarely be necessary. The condition of the kidney almost invariably can be better ascertained by pyelogram and intravenous urography, than can be done at operation when the surgeon has the kidney exposed. We have occasionally examined a kidney at the operating table which appeared normal, and had it not been for the pyelographic evidence the kidney could not justifiably have been removed. We also believe that kidneys occluded with tuberculosis are a probable source of infection dissemination, and should be removed regardless of the absence of symptoms. In general, then, when the pyelogram shows a destructive lesion of the kidney which is characteristic of tuberculosis, and particularly when the functional ability is diminished, and when the function of the opposite kidney is normal and the pyelogram is normal or only slightly impaired, we remove the worse kidney, followed by careful medical treatment.

ANÆSTHESIA

Some form of regional or spinal anæsthesia is the anæsthetic of choice, especially in renal tuberculosis. This is true because any inhalation anæsthesia may light up other tuberculous foci. A general anæsthetic will also impose an additional burden on remaining kidney. In our cases, average length of hospital days was less with regional than with inhalation anæsthesia (Table IV).

TABLE V.—ORIGINAL SYMPTOMS IN THIRTY-FOUR CASES

	Number	Per cent
Bladder symptoms	29	85.3
Pain in kidney	26	76.5
Cloudy urine	17	50
Hæmaturia	15	44
General symptoms	12	35.3

TABLE VI.—FINDINGS IN THIRTY-FOUR CASES

	Number	Per cent
Bladder appearance suggestive	25	73.5
Tubercle bacilli proved by guinea pig stain or potato culture	16	47
Able to catheterize both ureters	28	82.3
Functional test helpful	26	76.5
Pyelogram suggestive	28	82.3

SURGICAL PROCEDURE

In most cases, the kidney, with as much of the ureter as possible, is removed through the usual kidney wound. Beer suggests the possibility of producing a "tuberculous bacillæmia" by roughly handling the kidney prior to removal. Nephroureterectomy has been performed once in our clinic, but we believe that the necessity for this procedure is rare. A heminephrectomy for tuberculosis of the upper pole of the kidney was also performed once. This patient required a nephrectomy 1 year later because of a urinary sinus, at which time the remainder of the kidney also showed the presence of tuberculosis.

POSTOPERATIVE DATA

We have been able to trace 9 deaths. There were 3 postoperative deaths, as shown in Table VII, or slightly more than 3 per cent. The remainder have died from several months to years after operation. From Table III, it will be noted that the largest percentage of deaths occurred in the younger group of patients. Perhaps the infection is more acute in younger individuals or their resistance may be less than in older patients.

MEDICAL POSTOPERATIVE TREATMENT

One who treats tuberculosis must have a sympathetic understanding of this class of patient. He must be a solid support for them to lean on at all times, and one into whose sympathetic ears they may unburden their troubles. Dr. Wang of our clinic understands this psychology and always befriends his patients; consequently, he obtains complete co-operation and some excellent results. He suggests that the patient be informed of the exact nature of his disease in order to obtain his co-operation. The treatment is outlined at once, so that the patients realize the necessity for prolonged care. Their diet is placed on an almost

TABLE VII.—MORTALITY

Name, age	Operation	Mortality		Time after operation	Complications	Diagnosis		Anæsthesia
		Date of operation	Died			Guinea pig	Pathology	
C.C. 29	Left nephrectomy	11-20-23	2-24-24	3 mos.	Tuberculosis of bladder	+	+	Paravertebral
W.B. 42	Left nephrectomy	5-12-25	5-29-25	17 days	Uræmia, ureteral stricture	+	+	Paravertebral
H.K. 26	Right nephrectomy	12-12-22	1924	2 yrs.	None	+	+	Nitrous oxide gas, eth
R.M. 14	Right nephrectomy, ureterectomy	3-1-25	12-25	9 mos.	Rectal abscess, tuberculosis of ureter	?	+	Nitrous oxide gas, eth
E.R. 29	Left nephrectomy, ureterectomy	3-8-18	3-20	2 yrs.	Tuberculosis of ureter	?	+	Nitrous oxide gas, eth
L.P. 31	Left nephrectomy, ureterectomy	3-6-16	9-26	10½ yrs.	Pulmonary and ureteral tuberculosis	?	+	Nitrous oxide gas, eth
S.M. 14	Left nephrectomy, ureterectomy	5-11-15	6-5-15	25 days	Tuberculosis, ureter	+	+	Ether
S.B. 50	Left nephrotomy	8-7-31	8-11-31	4 days	Pulmonary embolus	+	?	Spinal
J.C. 46	Left nephrectomy (not done)	10-8-30	1932	14 mos.	Tuberculosis of bones, lungs, genitalia	+	+	Paravertebral

individual basis, for certain foods aggravate some and are beneficial to others. Wang (30) believes that most patients with genito-urinary tuberculosis should not be put in a sanatorium unless active pulmonary lesions are also present. Most public sanatoria are for patients with tuberculosis of the lungs, with no separate provisions for those with urological tuberculosis; and, usually, no one is particularly interested in the urological patient.

Rest, fresh air, quartz light, tuberculin, and medicinal therapy are employed, and the benefits derived are from the combination of these elements. The urological treatment consists in local urethral, bladder, ureteral, and renal treatments as indicated. Barnett, in discussing tuberculin therapy, states: "Never allow a skin or systemic reaction to occur without decreasing the dose. In other words, always be near a reaction or sometimes in one, but never on top of one." Warm gominal (29), 2 per cent, is instilled into the bladder twice a week for vesical irritations, and, combined with methylene blue by mouth, seems to be helpful to some patients.

In our most recent 34 cases, a lumbar sinus was present on leaving the hospital in 23, or 67.7 per cent. Many of these healed promptly with medical care. Wang (30) stated that all were closed in 8 months and an average of 4.2 months were required to heal these sinuses. In studying the cause of sinus formation, no conclusions could be reached since sinuses were present following nephro-ureterectomy, in old long standing disease of the kidney, and in early renal tuberculosis.

Pain in the remaining kidney was a distressing symptom for several months in many patients. This probably is due to the compensatory hypertrophy and increased blood supply to this kidney, with resultant tension of the renal capsule.

SUMMARY

The following summary is based on a review of the literature and a study of 97 patients who have had nephrectomy for tuberculosis:

1. Preclinical tuberculosis of the kidney is the stage between the time when the tubercle bacilli enter the kidney and the time when clinical evidence of extension to the bladder gives rise to symptoms. A few pus cells in the urine of an individual between 20 and 40 years (excluding venereal disease) should make one suspicious of renal tuberculosis.

2. The original symptoms complained of in 34 cases were: bladder symptoms, 85.3 per cent; pain in the kidney, 76 per cent; cloudy urine, 50 per cent; hæmaturia, 44 per cent; general symptoms, 35 per cent.

3. Diagnosis is established primarily by the pyelographic findings, with evidence of decrease in the renal function in advanced cases. We were able to catheterize both ureters in about 82 per cent of cases. The appearance of the bladder was suggestive of tuberculosis in 73 per cent of the patients studied. The finding of tubercle bacilli is not necessary for correct diagnosis in most cases.

4. Seventy per cent of the patients were between the ages of 20 and 40.

5. Intravenous urography is a distinct aid in some cases but cannot replace pyelography.

6. Bilateral pyelograms should be obtained routinely if the ureters can be catheterized.

7. Nephrectomy, followed by proper medical treatment, offers the only hope of arresting unilateral destructive renal tuberculosis. As much of the ureter should be removed as is possible through the kidney wound.

8. Nephrectomy should be performed in those patients manifesting a destructive lesion in one kidney with good function of the remaining kidney, where the second kidney can be shown pyelographically to be only slightly if at all affected. This is heroic, but offers the only chance of prolonging life, with a possibility of arresting the disease in the remaining kidney. Repeated cystoscopic examinations in an attempt to find tubercle bacilli from a pyelographically normal kidney, when the other kidney has a destructive tuberculous lesion, should be condemned.

9. Exploratory operations are, today, rarely if ever justifiable; more evidence should be obtained in most cases before surgery is undertaken than can be gained at the operating table.

10. Some form of regional or spinal anæsthesia should be used in preference to a general anæsthetic.

11. The mortality is higher in younger patients who develop renal tuberculosis.

12. The operative mortality was approximately 3 per cent.

13. A lumbar sinus was present in 23, or 67 per cent, of our most recent 34 cases when they left the hospital, all of which healed in an average of 4 months.

14. Pain in the remaining kidney following nephrectomy is common.

15. Medical care is an essential part of the treatment of renal tuberculosis and should be carried out by one thoroughly familiar with this work.

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TORSION OF THE SPERMATIC CORD[1]

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THIRTEEN years ago[2] I reported two instances of torsion of the spermatic cord. One was seen with Dr. William C. Quimby and the other was a patient of the late Dr. C. E. Porter. There was very little concerning this subject in the available textbooks and I found, after a complete review of the literature, that only 124 instances were on record. After this study it seemed to me that torsion of the cord must be a much more common occurrence than the records indicated. Since that time I have carefully questioned and examined each patient I have seen in whom there was complete or partial atrophy of the testicle. My conclusion is that torsion of the cord is not the clinical rarity formerly supposed and that atrophy of the testis following so called "orchitis" is in fact the end-result of torsion in a considerable percentage of instances.

The fact that there have been twice as many instances of torsion reported in the past 10 years compared with the entire literature preceding this time suggests a more common recognition of the condition rather than a more frequent occurrence.

In addition to reporting 6 additional personal observations together with 3 patients from the service of my colleague, Dr. Corbus, there are several reasons why this subject should be brought up again. Most patients with acute torsion of the cord are seen first by their regular physicians who, as a rule, do not consider this condition in making a differential diagnosis. The diagnosis of orchitis, epididymitis, or "acute hydrocele" is frequently made without proper correlation of history, symptoms, and physical findings. Since operative interference results in conservation of the testis only when resorted to in the first few hours after the onset of symptoms, it is necessary that the diagnosis be made at the earliest possible moment.

Torsion may occur at any age. It has been recognized shortly after birth and in one of my patients occurred at the age of 68 years. It probably occurs more frequently during childhood and adolescence, as indicated by the reported cases, although it is possibly more often correctly diagnosed during this period of life because the physician has less reason to err in the diagnosis of gonorrhœal epididymitis in these younger patients.

Torsion has been observed somewhat more commonly on the right side and in slightly more than half of the cases in incompletely descended testes. Here again it is possible that this recognition has not been because torsion occurs more commonly in undescended testes but because surgical interference has been more frequently advocated than in normally descended testes. The diagnosis of torsion was made during operation; the cause of the swelling was often not suspected before operation.

The most plausible explanation is that torsion is always brought about by contraction of the cremasteric fibers. There must be an abnormal attachment of the testis and a certain deficiency in make-up of the gubernaculum associated with a more or less capacious tunica vaginalis. This anatomical variation would not cause torsion, the twist itself resulting from repeated contraction of cremasteric muscle bundles, strands of which may be anomalous. Therefore, the degree of torsion, that is the number of half turns or full turns the cord undergoes, depends indirectly upon the freedom of the testicle to be rotated inside the tunica and directly upon the strength of the muscular contraction.

Torsion is said to come on most frequently during sleep but I have seen only one such instance in 11 cases. Strain of any sort, running, coughing, crossing the legs, etc., are mentioned as exciting factors. Gradual and repeated strain is more often noted in the recurrent cases; as coitus, and straining at stool.

At operation the pathological findings are characteristic. The cord above the torsion contains either dilated or flattened and partially obliterated spermatic veins, depending upon the extent of their occlusion in the twist. The spermatic artery is always greatly dilated but usually pervious. If the condition is an acute one, the surrounding tissue is œdematous; if chronic it is adherent and fibrous. In the acute cases the twist occurred from without inward in over two-thirds of the patients. In the recurrent cases no definite rule can be formulated. The extent of the twist varies from one-half turn to two full turns and the site of the twist is always in that free portion of the cord which, covered by tunica vaginalis, suspends the epididymis and testicle.

Incision into the tunica vaginalis reveals, in the early stages, more or less blood stained fluid while in the longer standing process old blood clot entirely fills the tunica. The remainder of the

[2] Surg., Gynec. & Obst., 1919, xxi, 580-584.

[1] Read before the North Central Branch of the American Urological Association, Detroit, Michigan, October 27, 1932.

cord below the twist, including the epididymis, is greatly swollen and bluish or purplish in color, while the spiral intersections produced by the twist are clearly seen. The testis itself may be only slightly enlarged or it may be treble the normal size.

On section the parts show intense engorgement, red, blue, or black, depending on the duration. In the longer standing cases there is a destruction of the testis, not a necrosis in the ordinary sense of the word but an aseptic death of the gland, which in process of time undergoes fibrification and consequent atrophy if left in the scrotum. In the recurrent cases any sharper attack than usual may cause lymph to be deposited in the tunica vaginalis and form adhesions anchoring the testicle. Microscopically there may be seen hæmorrhagic infarction; no organ left but merely old blood clot; or a diffuse interlobular hæmorrhage.

SYMPTOMS

The onset occurs with a varying degree of pain in the lower groin of the affected side. In the cases herewith reported 7 of the 9 felt pain more or less immediately after physical strain. In the acute cases which do not subside the pain becomes progressively more severe and localizes in the testis and lower cord. A characteristic diagnostic point is that swelling of the scrotal contents begins immediately and this swelling gradually increases for from 12 to 24 hours when it ceases. It is at this time that the severity of the pain is greatest. Careful observation will show that the swelling involves the cord only to the degree of the height of the twist, and the scrotum becomes diffusely reddened and tense. In the case of a testis lying high up in the inguinal canal the superficial structures of the groin assume this appearance. Another diagnostic feature, if seen in the first few hours, is that the swelling has the appearance of being drawn upward in the scrotum or inguinal canal, due to the shortening produced by the torsion.

When seen early the epididymis is normal in outline but is not in its usual relation to the body of the testis, being anterior or lateral rather than posterior. Toward the height of the swelling the outline of the testis and epididymis becomes obscured due to hæmorrhage into the tunica and generalized œdema of these tissues. There is very slight systemic reaction in the majority of cases, and the temperature, pulse, and leucocyte count do not seem compatible with the severity of the local condition. Nausea, vomiting, urinary frequency, weakness, and malaise may occur but are not part of the usual clinical picture.

If no attempt is made to untwist the torsion there is usually a gradual subsidence of all pain after 2 to 5 days, but the swelling and local tenderness persist for from 10 to 14 days. In 2 of my patients the pain was so severe 48 hours after the onset that generous doses of morphine failed to relieve it. The usual sequel is a gradual atrophy of the testicle due to fibrotic changes. The epididymis is much less affected in this atrophic process and in 4 of these cases has remained practically normal to palpation. Gangrene of the testicle has occasionally been reported but I have never seen torsion associated with active infection of the scrotal content.

DIAGNOSIS

In infants and younger boys orchitis, epididymitis, strangulated hernia, and acute inguinal adenitis must be differentiated from torsion. The average practitioner of medicine seems content with the diagnosis of "orchitis" for most acute or chronic swellings of the scrotal content. Strictly speaking *orchitis* as seen clinically is a relatively rare condition except for the orchitis accompanying mumps and the hæmorrhagic reaction resulting from direct trauma. Secondary orchitis, due to extension of infection from the epididymis, is usually so slight as hardly to justify the term except in rare cases in which the epididymal involvement is so intense as to block the circulation of the testicle by the peri-epididymal infiltration. Orchitis due to syphilis, whether it be the interstitial or gummatous type, is not an acute disease, involves the body of the testis and not the structure of the cord, and is never to be confused with torsion.

If the differential diagnosis will be analyzed on the acute onset, lack of findings coincident with epididymitis, either gonorrhœal or non-specific, exclusion of strangulated or incarcerated omental hernia, non-transilluminable scrotal content, and the finding of a reddened tense scrotum with elevation of the testis and limitation of the swelling to the lower inguinal canal, it is probable that fewer diagnoses of orchitis will be made and a higher incidence of torsion discovered.

PROGNOSIS

So far as recorded, torsion of the spermatic cord has never proved fatal. In recurring torsion the attacks usually continue with a resultant atrophy accompanied by intermittent neuralgic pain, unless an orchidopexy is performed. A few patients have had no recurrence after the first manipulation of untwisting the cord. The important feature of this condition is immediate diag-

nosis because in the acute cases operative detorsion in the first 3 or 4 hours will often result in the conservation of testicular tissue (Case 2). After 24 hours operation is only indicated to relieve pain or to eliminate the remote possibility of gangrene. In several instances seen after the severity of the pain had begun to diminish palliative treatment has been advised rather than operation; the prediction being of course that more or less complete atrophy would result (Case 8).

TREATMENT

1. *Detorsion* has been successful in a few cases of torsion in fully descended testes when seen very shortly after the onset of symptoms. It should, therefore, always be attempted in cases seen early but only in those in which the testis is outside the external abdominal ring. It is obviously impossible if the strangulation has existed long enough for engorgement of the testis to occur or for much fluid to accumulate in the tunica vaginalis.

Detorsion is accomplished by grasping the testicle between the thumb and second finger and slowly rotating on the vertical axis, trying first from within outward as the torsion most often found has occurred in a counter-clockwise direction. No force should be used and torsion should be continued until relief is felt or pain and resistance become so severe that it is obviously the wrong direction.

In one instance reported in the literature a patient with recurrent torsion was taught to perform detorsion successfully upon himself for many years. However, if after a successful detorsion has been accomplished, the condition recurs, an orchidopexy is indicated.

My most recent patient with this condition has been able to perform detorsion successfully for 4 months. His symptoms had been present for 3 months, and when first seen he had 50 per cent atrophy of the testis. There has been no increase in atrophy and so far he refuses orchidopexy (Case 9).

2. *Orchidopexy* should be performed in all cases in which it is deemed advisable to save the testis and in which there is no anatomical condition present that will interfere with the success of the procedure.

The usual transposition can be done on undescended testes with satisfactory results. In fully descended testes any operation that performs and accomplishes a fixation preventing recurrence will achieve a cure. A simple eversion and suture of the tunica vaginalis is usually sufficient.

3. *Orchidectomy*. In an adult where transposition of the undescended testis cannot be satisfactorily accomplished, or in any case in which necrosis, gangrene, or persistent circulatory obstruction is present, removal of the testis and the involved portion of the cord is indicated unless the patient has already passed through the period of pain and increased swelling. In this event, heat applied locally, and a week or 10 days of rest and a suspensory achieve relatively the same result as orchidectomy.

CASE REPORTS

(Cases given in abstract for purpose of brevity)

CASE 1. L. M., aged 13 years, a patient of Dr. Cor[illegible] and seen with him, appeared June 10, 1921, stating [illegible] after riding home from school on his bicycle he had [illegible] suddenly taken with a severe pain in the region of the r[illegible] testicle. The pain continued with increasing severity [illegible] night and when seen some 20 hours later had already be[illegible] to subside. The swelling involved the entire right scro[illegible] which was red and tense and symmetrical. There wa[illegible] tenderness or swelling above the external inguinal ring [illegible] the testis was definitely drawn up suggesting tors[illegible] Although not definite the epididymis seemed to be ante[illegible] to the body of the testicle. All other tests being negat[illegible], a diagnosis of torsion was made and immediate operation advised. The father preferred to wait and after 3 days of rest and hot applications the pain subsided and the swelling began to decrease. After 3 weeks the testis was replaced by a small soft mass with a normal epididymis felt anteriorly. This patient was seen in February, 1932, and practically no remnant of testis could be felt in the right scrotum, and although the epididymis was small it was of usual consistency.

Diagnosis: Torsion of right spermatic cord—atrophy. No operation.

CASE 2. C. S., aged 58 years, referred by Dr. Frederick Tice, was seen 2 hours after onset of severe pain in left testicle which came on after straining at stool. The testis was twice normal size, elevated toward the external ring, epididymis on external side of testis instead of posterior. Temperature and leucocyte count were normal and urine, prostate, and other tests were negative. A diagnosis of torsion of the cord was made and within 3 hours after the onset of symptoms the scrotum was incised and a rotated cord with one-half twist (180 degrees) was found just below the external ring. After detorsion and the application of hot pads for several minutes the circulatory flow seemed improved so the testis was replaced in the scrotum and several sutures served to anchor it in place. Operation was performed on September 3, 1927, and when last seen in December, 1930, the testis was normal to palpation and no further pain has occurred.

Diagnosis: Torsion of left spermatic cord.

Operative detorsion in 3 hours. No subsequent atrophy.

CASE 3. M. J., aged 36 years, came to the Washington Boulevard Hospital on October 12, 1927. He was a railroad brakeman and stated that on the previous day after uncoupling cars and jumping to the ground he was seized with a severe pain in the right testicle and had to be driven home. The pain increased constantly all night and on admission he required one-half grain of morphine to quiet him. The testis was drawn up in the right scrotum, was twice normal size, and very tender. The scrotal skin was reddened and slightly œdematous. There was no swell[illegible]

or tenderness above the right external inguinal ring. Because of a mucoid urethral discharge, which contained pus but no gonococci, the interne made a diagnosis of right epididymitis. Dr. A. R. Metz asked me to see the patient the next day because of an increase in swelling and pain. Temperature and leucocyte count were normal. A diagnosis of torsion of the cord was made and operation performed immediately. The cord just below the external ring was twisted one and one-half times (540 degrees) and the testis was completely replaced by blood clot, and disintegrated reddish-brown material. The cord was severed 2 inches above the twist and both testis and cord removed *en bloc*.

Diagnosis: (Dr. E. R. LeCount) *Necrosis of, and hæmorrhage into, right testicle due to torsion of the cord.*

An interesting feature of this case, not previously seen mentioned by me, was a subsequent claim for damages against the company on the contention of injury compensible in line of duty. Settlement was made out of court.

CASE 4. A. K., aged 31 years, employee of an X-ray company, was referred by his employer on April 4, 1927. He stated that on April 1, while adjusting some equipment he had fallen from a ladder about 5 feet from the ground. A severe pain in the right groin caused him to double up and within an hour the right scrotum was swollen and tender. He returned to Chicago from the small town in which he was working and remained in bed, applying heat to the scrotum, for 2 days. When first seen the pain was very severe. Temperature, leucocyte count, and all other tests were normal. The right scrotum was three times normal size, tender, and non-transilluminable. The swelling extended up to the external ring and was pear-shaped at this point. A diagnosis of torsion was made and immediate operation performed. The cord was twisted almost one complete turn (345 degrees) and there was an apparent dry gangrene of the tissues within the tunica. The twist was a spiral one extending from the external ring and terminating just above the globus major. The cord and testis were removed.

Diagnosis: Torsion of the right spermatic cord. Orchidectomy.

This patient returned on October 28, 1928, stating that he had had fleeting pains for several months in the left testicle. Examination revealed no evidence of torsion but a moderate scrotal varicocele. Having read up on this subject the patient was in fear of torsion on the left side and demanded an orchidopexy. This was done and examination in October, 1932, shows a normal testis free from pain.

CASE 5. M. H., aged 68 years, Jewish rabbi, was seen with Dr. Dolnick on March 8, 1929. Four days previously on stepping out of the bath tub the patient felt a sudden severe pain in the right groin and scrotum and immediately noted a slight swelling. The pain continued to increase and the swelling progressed until the scrotum was four times normal size. A diagnosis of strangulated inguinal hernia was made but on hospital examination the temperature, leucocyte count, and bowel movements were normal and the diagnosis was changed to orchitis. The pain was so severe that morphine would not relieve it and the patient had to be strapped in bed and attended by a burly male nurse to prevent attempts at throwing himself out of the window. On examination (4 days after onset) the swelling extended to the external inguinal ring at which point it was pear-shaped. The skin of the scrotum was blue but not œdematous. No outline of epididymis could be felt. All other tests being negative a diagnosis of torsion was made and operation performed in a few hours. The cord was twisted one and one-quarter times (450 degrees) and a large sclerotic spermatic artery could be seen ineffectually pumping at this point. The testis was replaced by 300 cubic centimeters of bluish black, bloody fluid with a few strands of testicular tissue floating about.

Diagnosis: Torsion of right spermatic cord. Destruction and hæmorrhage into testis. Orchidectomy.

CASE 6. (Reported through courtesy of Dr. B. C. Corbus.) Boy, aged 11 years, referred by Dr. L. Hagen on March 6, 1928. Two days previously he had fallen from his bicycle and within an hour experienced pain in both testicles. There had been no direct trauma to the scrotum and there was no bruise or ecchymosis. Both testicles were twice normal size, drawn high up in the scrotum with pear-shaped swelling terminating at the external ring. The vasa on either side were normal and no nodules could be felt. All other tests were negative, temperature and leucocyte count normal. A diagnosis of bilateral torsion of the cord was made and immediate operation advised in the hope of saving some testicular tissue. Another consultant, however, disapproved this procedure and diagnosed the case as one of acute tuberculosis of both epididymides. The swelling and pain persisted for 2 weeks and then began to subside. The boy was taken to California and given several months of outdoor treatment. During this time the swelling completely disappeared and examination in October, 1932, by Dr. Hagen shows complete atrophy of both testes with normal epididymides and vasa. The boy now shows evidence of testicular deficiency in his development. There now seems no doubt about the accuracy of the diagnosis of bilateral torsion and it seems too bad that operative interference was denied this boy even if the chance of retaining some testicular activity was slight.

CASE 7. (Reported through courtesy of Dr. B. C. Corbus.) J. R., aged 4 years, was admitted to the Evanston Hospital on December 10, 1929. His parents stated that 36 hours previously he had complained of sudden severe pain in left scrotal region after coming in from the playground. When put to bed the pain disappeared and did not recur until the middle of the night, when the testis began to swell and the pain returned for several hours. Twelve hours prior to admission the pain recurred with constant severity and the scrotum became reddened and more swollen. On admission the child was in severe pain, the scrotum red and tense, and a swelling extending to the external ring. Temperature 100.4 degrees F., pulse 120, respiration 20, normal urinalysis and leucocyte count. Physical examination was negative in every respect except for the left scrotal swelling and the right testis retained in the inguinal canal. A diagnosis of torsion was made and immediate operation performed. The left spermatic cord was twisted just below the external inguinal ring one and one-half times (480 degrees). This was untwisted after freeing the testicle and after 10 minutes of hot applications the circulation seemed re-established. The testis was returned to the scrotum and fixed in place. Recent examination shows an atrophy of about 70 per cent, normal epididymis.

Diagnosis: Torsion of left spermatic cord. Operative detorsion; orchidopexy. Subsequent partial atrophy of testis.

CASE 8. S. R., aged 24 years, referred by Dr. Broyde on January 12, 1932. Five days previously, after carrying an armful of heavy law books, he had a sudden knife-like pain in the right groin. The pain continued for 48 hours and during this time the right scrotum gradually became larger, more tender and reddened. Morphine and hot applications were given for the next 48 hours to aid in quieting the pain. When I saw the patient the swelling was in the upper right scrotum terminating in a knob-like pro-

tuberance just below the external ring. The structures above were normal. Temperature and leucocyte count had been normal. Prostate and urine were normal. The diagnosis of torsion of the right spermatic cord was made and no further treatment except rest advised. An atrophy of the testis was predicted. On February 12, 1932, the patient had normal scrotum, epididymis, and vas, and almost complete atrophy of the right testis.

Diagnosis: Torsion of the right spermatic cord; no treatment; subsequent atrophy of the testis.

CASE 9. H. R., aged 39 years, referred by Dr. Fisher on April 12, 1932, with the complaint that for 3 months he had had intermittent attacks of pain in the right testis and groin accompanied by transitory swelling and elevation of the right testis. There was no history pertinent to this complaint, no previous venereal disease, and no sign of hernia. The patient had never had mumps. Examination disclosed a moderate swelling of the right scrotum and the testis high up with epididymis anterior to the body of the testis. By grasping the testis between the thumb and middle finger and rotating in a clockwise direction the epididymis was placed posteriorly and the swelling and pain subsided in a few minutes. For economic reasons the patient refused orchidopexy and after instructions as to the procedure of performing detorsion himself, while sitting in a tub of hot water, he has successfully relieved the condition on 6 occasions. When last seen on October 3, 1932, he had 50 per cent atrophy of the right testis and, although again advised to have the testis fixed in place surgically, feels satisfied with the present condition.

Diagnosis: Recurrent torsion of the right spermatic cord; successful non-operative detorsion; partial atrophy of testis.

SUMMARY

Torsion of the cord is not the clinical rarity previously implied by isolated case reports. Nine additional cases are cited, 5 of which were verified at operation, 3 by the findings and subsequent course, and 1 an obviously recurrent type with gradually developing atrophy. Orchitis, as a clinical entity, except in association with epidemic parotitis is an uncommon condition and the term should not be loosely applied to explain undifferentiated scrotal swellings. Bearing in mind the characteristic sudden onset of torsion with the more or less typical local findings will enable an early diagnosis with immediate operative detorsion which is necessary in most acute cases if testicular tissue is to be conserved.

BILATERAL LOBECTOMY

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LOBECTOMY for intractable suppurative bronchiectasis is gaining favor. Most surgeons, and not a few internists, agree in theory at least that removal of the affected lobe is the reasonable and desirable method of attacking this hopeless and distressing disease. The high risk of the operation has prevented its performance in all but the most desperate cases, and has caused many surgeons to cast about for less dangerous substitutes. None of the substitutes, however, have given as many complete and satisfactory recoveries in the patients surviving operation as has removal of the suppurating portion of the lung. Many patients that have submitted to thoracoplasty, and other indirect methods of attack, as well as many that have been operated upon by cautery pneumotomy, have had to undergo tedious and trying periods of treatment from which the survivors have emerged, some of them crippled, some not, but not many really cured. It is not surprising, therefore, that some surgeons, who frightened by their operative deaths abandoned the operation, have bent their efforts toward lessening its danger, and have returned to it again and again, while other pioneers, notably among them Lilienthal, have remained unappalled and held steadfastly to a method that seemed to them to be sure of ultimate success.

The dangers of lobectomy are diminishing. In 1925 Lilienthal, who has had a wider experience than anyone else, reported 34 lobectomies for bronchiectasis and allied suppurations with 21 deaths—a mortality of 62 per cent.[1] In June of this year Ballon, Singer, and Graham report a collected series of 212 lobectomies by various operators with a mortality of 34 per cent.[2]

Although the death rate has been halved in the last 7 years intervening between Lilienthal's textbook and the recent collected report, it is still too high to make the operation one to be advised for any but hopeless sufferers. As the authors say: "Even if we take only the more recent and the more favorable figures we still find that a patient with bronchiectasis who submits to a lobectomy runs about a 15 or 20 per cent risk of dying because of the operation, and that if he recovers from the operation he has only about a 65 per cent chance of having a thoroughly satisfactory result with solid healing of the wound." This is true, yet the halving of the mortality in the last 7 years is promising, and the recent successful series of many operators—Alexander, Brunn, Sauerbruch, Shenstone, and others—give reason to hope that bronchiectasis may yet prove to be a disease curable without too great a risk.

Surgical efforts have hitherto been confined to more or less unilateral bronchiectasis. Bilateral disease has usually been barred from active intervention. Krampf,[3] however, in a recent communication from Sauerbruch's clinic states that we should consider abandoning our conservative attitude toward bilateral bronchiectasis; he states that one side is often worse than the other and that surgical attack on the worse side should be considered.

This is not the place for an extensive discussion of lobectomy; however, the following report of a bilateral lobectomy in a patient observed over a period of 8 years may serve to show its possibilities.

On February 8, 1924, Dr. Frank Sheehy was so kind as to ask me to see Miss Dorothy F—— with him. She was then 16 years old. She had coughed and expectorated ever since she could remember, certainly since the age of 5 years. She had the measles as a child, but it is not certain that her cough dated therefrom. She had pneumonia in 1917 which lasted 3 weeks. She had a severe attack of influenza during the 1918 epidemic; her expectoration ceased during this attack. One of her three brothers has a bronchiectasis, two others and a sister are well. Her parents had no lung trouble.

On first examination she was a rather pale child with numerous acne postules and their scars. Her fingers were not clubbed. The lower left side of the chest lagged a little. There was a triangular area of dullness over the left base next the spine, with diminished breath sounds. The sputum was thick, green, foul, and occasionally contained a little blood. It amounted to 180 cubic centimeters per day and 120 cubic centimeters at night. Repeated examinations failed to reveal tubercle bacilli, fungi, or other specific organisms. Cultures gave a growth of almost pure streptococci in short chains. X-ray films demonstrated a triangular shadow lying behind the heart, and for the greater part covered by it.

We thought that this shadow might represent a localized empyema to which bronchiectasis might be secondary.

On February 16, 1924, about 3 inches of the eighth, ninth, and tenth ribs were resected. The pleura was thickened. No pneumothorax resulted until aspiration of the area corresponding to the triangular shadow was attempted. No pus was found. The pleura was opened. There were a few fine elastic adhesions between the base of the lung and the diaphragm. There was no empyema; the

[1] Thoracic Surgery. Philadelphia and London. Vol. ii, p. 216.
[2] J. Thoracic Surg., 1932, i, 540.
[3] Payr-Kuettner. Ergebn. d. Chir. u. Orthop., vol. xxiii, p. 652.

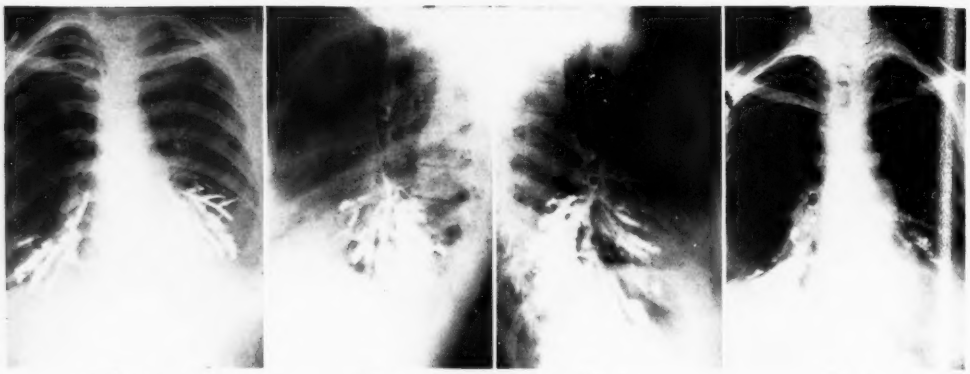

Fig. 1. Fig. 2. Fig. 3. Fig. 4.

Fig. 1. February 21, 1928. Bilateral bronchiectasis before lobectomy. Large tubular dilatations of both lower lobes filled with iodized oil. Anteroposterior projection.

Fig. 2. February 21, 1928. Bilateral bronchiectasis before lobectomy. Large tubular dilatations of both lower lobes filled with iodized oil. Right lateral projection.

Fig. 3. February 21, 1928. Bilateral bronchiectasis before lobectomy. Large tubular dilatations are noted in both lower lobes which are filled with iodized oil. Left lateral projection.

Fig. 4. July 8, 1932. Bilateral bronchiectasis after removal of both lower lobes. Some dilated bronchi still remain in the right middle lobe from which the patient's sputum probably comes.

triangular density corresponded to a doughy infiltrate which lay posteriorly in the substance of the left lower lobe. Its base measured about 2½ inches across and lay on the diaphragm. It ran up about 4 or 5 inches toward the hilum. A needle introduced into it withdrew a little pus. The infiltrated area was attached to the chest wall by a gauze pack, sewed over it, and the chest was rapidly closed.

An effusion formed in the chest which splinted the lung and reduced the sputum first to 50 cubic centimeters, then to nil. In view of the cessation of expectoration, the lung was not opened. Some 6 weeks after operation the patient's temperature rose and the effusion became purulent. A large amount of pus was discharged from the incision.

The empyema healed rapidly, but as it healed and the lung expanded, cough and expectoration returned. In August, 1924, 6 months after operation she had bubbling râles over both bases and I thought that the bronchiectasis was bilateral.

A year after the first operation, in February, 1925, she was admitted to the San Francisco Hospital. On February 12, 1925, the old scar was excised, pieces of two ribs were resected, and the adherent lung cauterized. Several cavities were opened. The fistula closed very rapidly; the sputum remained undiminished; it was thought to come from the right side. She was discharged April 11, 1925, with 125 cubic centimeters of sputum and a wound that was still open but drained very little. The wound closed in May, 1925.

X-ray pictures of March 16, 1925, with iodized oil disclosed dilatation of the right basal bronchi. In November, 1925, she had no more fever but expectorated 5 or 6 ounces of sputum daily.

In February, 1928, I saw her again. She had been at Banning in southern California taking sunbaths. She had lost considerable weight, had intermittent attacks of fever, swelling of the joints, and severe headaches. In September, 1929, she had gained weight, but was otherwise worse. The sputum measured 6 or 7 ounces per day. Bronchoscopy on February 24, 1930, revealed much secretion from the right bronchi, less from the left. The larger bronchi were anatomically normal. In October, 1930, the ethmoid sinuses were drained by Dr. W. B. Smith. This improved her headaches, but not her expectoration. On March 3, 1930, 3½ inches of the seventh, eighth, and ninth ribs were resected, the wound was packed with gauze, and the chest closed. On March 21, the wound was reopened and a saucer shaped portion of the adherent pleura and lung removed with a Percy cautery. A number of bronchi were opened from which air came, but no pus. This opening, like that in the left side, healed rapidly. The sputum was undiminished, and finally a year and a half later, on September 26, 1931, a right lower lobectomy was decided upon. My notes read:

An incision was made in the old scar and the pleural attachment of the bronchial fistula, and therewith the right lower lobe exposed. At this juncture the patient stops breathing and becomes very blue; however, upon her giving a few breaths, operation is proceeded with and the right lower lobe, which is much shrunken but riddled with bronchiectatic cavities, rapidly freed of its attachments. With some trouble, through the small chest opening, three or four right angled clamps are placed on the hilum and each of them tied with a black silk ligature which is left long and led out through the wound. The lower cavity is packed with gauze soaked with mercurochrome. The bottom of the right middle lobe, which comes to view as the anæsthetist dilates it, is pink and air-containing.

Following operation the rectal temperature rose to 103 degrees and the pulse to 110; her respiration reached 30 some days later. Temperature and pulse rate receded to normal in the course of 2 weeks. At no time did the patient appear alarmingly ill. She stated that her discomfort was far less than after her previous cautery operations. The ligatures came away about 2 weeks after operation. The patient was out of bed on the nineteenth day and was discharged on the twenty-third day. At the time of her discharge she had normal temperature; her sputum averaged 30 to 45 cubic centimeters per day. In the next few months she continued to have intermittent attacks of fever and

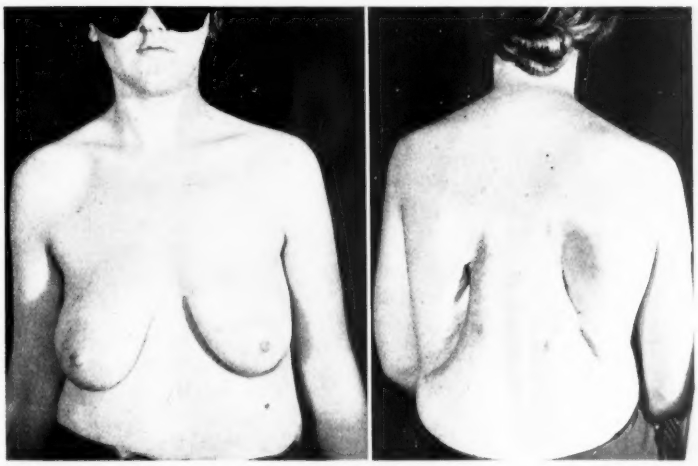

Fig. 5, left. Patient after bilateral lobectomy.
Fig. 6. Patient after bilateral lobectomy. The left sided opening has entirely closed since this photograph was taken.

the sputum rose to 3 to 4 ounces per day. There was considerable discharge from the bronchial stump, but the fistula closed spontaneously about January, 1932, and has remained closed since. Sputum and intermittent attacks of fever continuing, it was decided to remove the left lower lobe over which râles could constantly be heard. This was done January 26, 1932. My notes read:

In a girl who, after right sided lobectomy last August, continued to have sputum which, from the physical signs and from the X-ray examinations, doubtless came from the left lower lobe, removal of this lobe was decided upon in the hope of ridding her of her profuse expectoration. This lobe had been cauterized according to the method of Graham years before.

The old scar was therefore opened under satisfactory local and gas anæsthesia. The adhesions between the parietal chest wall and the site of the operative defect were separable without difficulty. Those to the diaphragm and those in the entire lobar fissure were so dense that, in separating them, a hole was burnt with the cautery into the diaphragm, which was recognized and immediately repaired with chromic gut. The hilum having been reached, two large Wertheim clamps were applied to it, tied about with silk ligatures on a needle, the clamp reapplied and the lobe removed above the clamp with the cautery. It was carnified and contained large bronchiectatic cavities from which thick pus exuded. The resulting cavity was packed with a Mikulicz tampon and plain gauze.

Again she made a good recovery. Her temperature rose to 102 degrees after operation and the pulse to 110. By the tenth day the temperature was fluctuating between normal and 100, and the pulse between 70 and 90. The respiration at no time exceeded 28 per minute. She was out of bed on the twenty-second day after operation and was discharged from the hospital on the twenty-sixth day.

On July 7, 1932, she stated that for about a month after the last operation cough and expectoration had practically ceased. She then caught a "cold" and had much pain in the head, especially on the right side. She had a discharge from the nose. Following this cough and sputum increased so that it now reaches 120 cubic centimeters per day.

On examination the bases of both lungs bulged considerably through the defects in the chest wall. The right chest had remained closed. On the left there was a minute spot of granulation tissue through which about ½ cubic centimeter of pus came when she coughed, but no air. There was a strip of bronchial breathing at both bases between the two scars, but no râles were to be heard. A pressure bandage was applied to the bulging scars and held in place by a few turns of plaster of Paris. This greatly diminished the cough and expectoration which now amounts to 60 cubic centimeters daily. A celluloid and metal splint has been made to take the place of the plaster dressing. The vital capacity on August 15, 1932, measured 1,588 cubic centimeters. The patient attends to her housework and is not dyspnœic on ordinary exertion.

REMOVAL OF VERTEBRAL BODIES IN THE TREATMENT OF SCOLIOSIS[1]

HERMAN LEROY VON LACKUM, M.D.,[2] AND ALAN DE F. SMITH, M.D., F.A.C.S., NEW YORK

THE impossibility of correcting by ordinary methods lateral curvatures caused by hemivertebræ led to the attempt in July, 1924, to remove the anomalous wedge-shaped body and its arch. Because growth of the vertebral column is not checked by the spine fusion operation, it is believed that this procedure alone could have no effect upon the progress of such a curve, and that the latter would continue to increase so long as the hemivertebra continued to grow. It also is self-evident that jackets or other apparatus could have no influence whatever upon such a deformity. The application of the operation of partial vertebral removal to the treatment of certain advanced cases of scoliosis not due to hemivertebræ, in which marked structural deformity prevented correction by ordinary means, next suggested itself. From July, 1924, to June, 1928, 10 patients, 5 with curvatures caused by hemivertebræ and 5 with ordinary scoliosis, were operated on for removal of vertebral bodies.

The ages of the patients at the time of operation varied from 1½ to 22 years, with an average of 11½ years. The hemivertebra group were younger than the scoliosis patients, the former averaging slightly more than 6 and the latter 17 years. Because it was believed at first that the best results could be obtained only in young children before severe deformity had become established, the first 2 patients were operated on at a very early age, 3 years and 17 months, respectively. It soon was appreciated that children of this age could not well tolerate an operation of this severity and no further cases were undertaken at less than 6 years.

The plan of operation in the earlier cases was to approach the vertebra through a dorsal midline incision over the spinous processes, or one parallel to and a short distance from the midline. The posterior arches were exposed subperiosteally and the spinal muscles were divided transversely on one side at the level of the vertebra to be removed and retracted laterally. In the dorsal region the posterior portion of three ribs, including the one above and that below the intended vertebra, were resected with the transverse processes. This gave a rather poor exposure of the side of the vertebral body with its pedicle. The lamina and pedicle were removed first and then part of the body. The latter usually had to be gotten out piecemeal with a curette or gouge. This procedure was difficult because of the inadequate exposure but chiefly due to the severe hæmorrhage. In the cases thus operated on the bleeding was profuse and in the majority there was severe or moderately severe shock, making it necessary to give transfusions to several of the patients after operation. The second patient operated on, the youngest in the series, succumbed to shock one hour after operation. As soon after removal of the vertebral body as the patient's condition allowed a traction jacket, with antero-posterior hinges and a turnbuckle, was applied and the greatest possible correction was obtained. A Hibbs spine fusion operation was then done.

The third patient upon whom operation was proposed was a boy of 6 years with a hemivertebra between the third and fourth lumbar on the left side, causing a curve in that direction. This suggested an easier approach to the vertebral body by an incision through the flank, behind the peritoneum. It was made from the twelfth rib at the outer border of the sacrospinalis muscles, downward and forward through the dorsolumbar fascia to the crest of the ilium, from the central third of which the muscles were divided. The peritoneum was pushed forward, thus giving a good exposure of the lumbar bodies on that side. It then remained simply to ligate the lumbar vessels above and below the body and to remove the latter with a chisel as far as its junction with the pedicle. There was very little bleeding and no shock. The whole procedure was accomplished in less than one hour.

Twelve days later, through a dorsal midline incision, the posterior arch, together with those adjacent to it, was exposed by subperiosteal dissection, and the arch of the hemivertebra was removed. Although not extremely difficult, this second stage of the operation resulted in more bleeding and was not as easy as the first. The hemivertebral arch usually is united to the one above or below, and sometimes to both, which adds another obstacle to its excision after removal of the body. Bridges of bone were turned up and down from the adjacent laminæ in such manner that they would interlock when the laminæ were approximated.

After both stages of the operation the patient was placed in a posterior shell of plaster previously

[1] From the clinic of the New York Orthopædic Dispensary and Hospital.

[2] Dr. von Lackum met with an untimely death in an airplane accident on June 30, 1928. All of the operations for removal of vertebræ reported in this paper were performed by him. The operations on these patients for spine fusion, etc., after that date were performed by his colleagues.

prepared. A jacket was applied under light traction 9 days after the second operation, and in this a lateral hinge and turnbuckle were incorporated by means of which the jacket was wedged and the space left by removal of the hemivertebra was closed. Fusion occurred in this case without further operation, but in a similar case it was necessary to do a fusion operation later.

Done in this way, the operation is comparatively easy and is free from hæmorrhage and shock. It is applicable only to cases of hemivertebra in the lumbar region, and it is important to note in this connection that the only successful cases among these ten were two with lumbar hemivertebræ and one of scoliosis in the lumbar region. The difficulty of exposure and the severe hæmorrhage encountered in the dorsal area of the spine make the operation impracticable there.

One patient, a girl 14 years old, with a severe right dorsal curve, had a pneumothorax following removal of part of the ninth dorsal body. This was the only case in which the pleura was damaged. A child 6 years old, with a curvature caused by a hemivertebra and who also had an extensive spina bifida in the lumbar region and partial paralysis of the lower extremities, showed an increase in the paralysis and evidence of spinal cord pressure after operation for removal of the twelfth dorsal body.

The results of the operations in the 10 cases may be summarized as follows: Two patients with hemivertebræ between the third and fourth lumbar were corrected to the point where their spines were straight and there was no apparent curvature. In the first the curve was reduced from 31 to 9 degrees, and in the second it was overcorrected from a left curve of 37 degrees to a right curve of 7 degrees.[1] One 22 year old patient with a marked left lumbar curve was corrected from 30 degrees to 6 degrees by removal of portions of the first and second lumbar bodies. Two other patients obtained substantial reductions in their lateral curves, but this was offset by the occurrence of a kyphosis and failure of fusion of the spine at the site of operation. The other cases either were not corrected beyond the condition before operation or subsequently progressed still further. These were all dorsal curves and the failures are attributable to inability to complete the removal of the body, or portion of the body, because of hæmorrhage. One patient died of shock. The average period of observation after operation was 4½ years.

[1] The method of measuring the degree of the curvature in scoliosis is described by Ferguson in "The study and treatment of scoliosis." South. M. J., 1930, xxiii, 116.

Pseudarthrosis, or a defect in the spine fusion at the site of operation, occurred in 6 of the 9 cases. An operation for repair was done twice in 2 cases and once in 2 cases. The 2 other patients refused to have another operation. One complains of tenderness at the pseudarthrosis. The other has no symptoms.

A kyphotic deformity developed at the point where the vertebral body was removed in 7 of the 9 patients. It was marked in 5, moderate in 1, and slight in 1. This was in the dorsal cases and was due to anterior rather than the desired lateral collapse.

A brief history of the cases follows:

CASE 1. J. W., No. 53,444, a boy, was first seen in the out-patient clinic of the New York Orthopædic Hospital March 28, 1922, at age of 8 months. Physical examination was negative except for a marked left lateral curve of the spine in the lower dorsal region and a right lumbar curve. X-ray examination showed a hemivertebra of the eleventh dorsal on the left side and a hemivertebra of the second dorsal, right. Attempts to correct the curve by plaster jackets preliminary to spine fusion were made in 1923. These were unsuccessful and the curvature increased, associated with a kyphosis. At the age of 3, on July 15, 1924, an operation was performed for removal of the eleventh dorsal hemivertebra. Through a dorsal midline incision the lamina and pedicle of the eleventh dorsal were removed, after which the body on the left side was exposed between the nerve roots, and excised. The Hibbs spine fusion operation was then done between the ninth and tenth dorsal and the twelfth dorsal and first lumbar arches, but not between the tenth and twelfth, in order that the curve might be corrected. Subsequently X-ray examination showed that the eleventh dorsal hemivertebra had been removed and that there was a 52 degrees' curve from ninth dorsal to second lumbar. By means of plaster jackets the curve was reduced to 20 degrees, and on October 14, 1924, a second fusion operation was done to unite the arches of the tenth and twelfth vertebræ between which the eleventh had been removed. A jacket was reapplied and he was maintained in plaster until July, 1925. In September the curve had increased to 40 degrees and the patient was readmitted and operated on. A wedge was removed from the left side of the tenth dorsal vertebra. This body was approached through a midline incision. Failure of fusion was found between the arches of the ninth, tenth, and twelfth dorsal. A wedge of bone with base to the left was removed at each site of pseudarthrosis. Two weeks after operation a plaster jacket with head piece was applied under traction. The patient was readmitted to the hospital April 27, 1927, for repair of pseudarthrosis. A jacket was applied which caused a pressure sore due to a kyphosis which had developed at site of operation. The operation was performed on September 23, 1927. A narrow crack was found at the apex of the kyphos and another at the upper end of the fusion area. These were repaired. In April, 1931, the left curve measured 50 degrees and the kyphos 90 degrees. Fusion appeared to be solid.

CASE 2. L. F., No. 82,762, a girl, was brought to the clinic of the New York Orthopædic Hospital when 10 months old because of a deformity of her back noted 2 months previously. She was a well developed, healthy baby with a moderate left lateral curve in the dorsolumbar region. X-ray examination showed a left hemivertebra of the twelfth dorsal. She was admitted to the hospital

Fig. 1.

Fig. 1. Case 3. Roentgenogram before operation, showing left hemivertebra between third and fourth lumbar vertebræ.

Fig. 2.

Fig. 2. Case 3. X-ray picture taken in hinged jacket after operation. Hemivertebra has been removed.

Fig. 3.

Fig. 3. Case 3. Final result 2 years after operation

and operated on for removal of the hemivertebra on December 7, 1925, at the age of 1 year, 5 months. Through a midline incision the posterior arch of the hemivertebra was exposed subperiosteally. The upper part of the lamina was removed and the lateral and anterior aspects of the body were exposed. The hemivertebral body and part of the body of the first lumbar were removed with a curette and a bivalved plaster was applied. The patient recovered from the operation without shock. X-ray examination after operation showed that the hemivertebra apparently was still present, removal evidently having been incomplete. On February 15, 1926, a second operation was undertaken for removal of the hemivertebra. This was done through the scar of the previous operation. Because of the great difficulty in exposing the residue of the hemivertebral body the one below this (first lumbar) was removed piecemeal. Bleeding made the operation difficult but the patient left the operating table in fairly good condition. A hypodermoclysis was given immediately. She suddenly went into shock and died 1 hour after the operation.

Case 3. B. B., No. 99,256, a 6 year old boy, was first seen in February, 1927. Eight months previously the family physician noted a slight curvature of the spine. An operation for pyloric stenosis was done at 4 weeks of age. He was a well developed and nourished boy. Posture was poor and there was a left lateral curve in the lumbar region. X-ray examination revealed a left hemivertebra between the third and fourth lumbar, and a central cleft through the bodies of the fifth, sixth, and eleventh dorsal vertebræ. It was decided to remove the lumbar hemivertebræ in two stages, the body through a lateral incision in the flank and the posterior arch through a dorsal midline incision. The first stage was done on April 9, 1927. The hemivertebra was united to the body of the fourth lumbar and was chiseled free. It was then removed with the disc between it and the third lumbar. A previously prepared plaster shell was applied. There was no postoperative shock. The second stage was done on April 21, 1927, 12 days after the first. The lamina, transverse process, pedicle, and small remaining part of the body of the hemivertebra were removed and a Hibbs fusion operation was done between the third and fourth lumbar vertebræ. A posterior plaster shell was applied. There was no shock and the incisions healed by primary union. A jacket with lateral hinge was applied under slight traction 9 days after operation. The jacket was wedged and the space left by removal of the hemivertebra was thus closed, bringing the third and fourth lumbar bodies in contact. The patient was discharged from the hospital, in a bent plaster jacket on May 20. This was removed in September, 1927. X-ray examination in May, 1932, showed a left lumbar curve of 9 degrees instead of the 31 degree curve present before correction. Physical examination showed that the spine was straight and the shoulders and pelvis level. Two brothers of the patient have vertebral anomalies in the form of cleft bodies and arches of several dorsal vertebræ.

Case 4. S. D., No. 97,213, a 17 year old girl, applied for treatment of a lateral curvature of the spine at the New York Orthopædic Hospital on January 18, 1927. The deformity first was noticed when she was 12 years old and was treated for years with exercises, massage, braces, and corsets, in spite of which it constantly increased. She had a marked right dorsal curve of 46 degrees, extending from the sixth dorsal to the first lumbar vertebra, with the apex at the tenth dorsal. There was a compensatory left lumbar curve of 34 degrees. Attempts to reduce the curve by jackets applied in the dispensary failed. She was admitted to the hospital and on October 26, 1927, the pedicle and about one-third of the body of the tenth dorsal were removed through a six inch incision to the right of the midline. The operation was very difficult because of profuse hæmorrhage. Her condition after operation was poor and the pulse was rapid and irregular. A transfusion of 700 cubic centimeters

Fig. 4.

Fig. 5.

Fig. 6.

Fig. 4. Case 6. Roentgenogram of hemivertebra between second and third lumbar vertebræ showing the condition before operation.

Fig. 5. Case 6. Roentgenogram taken immediately after operation and before correction, showing removal of the hemivertebra together with a large part of the third lumbar body.

Fig. 6. Case 6. X-ray picture 3 years after operation, showing overcorrection of the curve and fusion of the bodies.

of blood was given in the evening, after which the patient's condition was much improved. X-ray examination 3 days after operation showed the dorsal curve reduced to 36 degrees. A wedging jacket was applied by means of which the curve was further corrected to 24 degrees.[1] On December 7, 1927, 2 months after the first operation, a spine fusion of the ninth, tenth, and eleventh dorsal was done through a window in the jacket. She was discharged from the hospital, in a bent jacket, on March 10, 1928. An X-ray examination on June 6, 1930, showed that the right dorsal curve had increased from 24 to 61 degrees and that there was a marked kyphos with the apex at the tenth dorsal. In January, 1932, the patient was in good general health and had no pain or fatigue in her back. She was employed as a dentist's assistant. The kyphos had increased markedly since 1928 but more rapidly in the last 18 months, indicating that a pseudarthrosis was present in the fusion area. She refused to have this repaired.

Case 5. T. C., No. 51,871, a girl 9 months old, was admitted to the out-patient clinic of the New York Orthopædic Hospital on January 6, 1922. She had a right dorsal, left lumbar curve and X-ray films revealed a hemivertebra on the left side between the second and fourth dorsal and another at the twelfth dorsal on the left side. There was a spina bifida from the first lumbar to the sacrum. On admission to the ward in January, 1928, she had a fixed left dorsolumbar curve of 50 degrees, with marked tilt of the pelvis, and a valgus deformity of the left foot and equino varus of the right, caused by the spina bifida. Following a blood transfusion to improve her general condition, an attempt was made on February 24, 1928, through a dorsal incision, to remove the arch and body of the twelfth dorsal hemivertebra. Great difficulty was encountered because of hæmorrhage, and after removal of the arch and pedicle the excision of the body was abandoned. Two transfusions were given after operation. A second attempt was made to remove the body on June 4, 1928, through a left lumbar incision. Again, profuse hæmorrhage occurred but the hemivertebra and about half the body of the first lumbar were removed. The wound was packed for 48 hours. Following operation the paralysis of the lower extremities, which was present before operation, was greatly increased, and retention of urine developed 4 days later. A wedging jacket after operation failed to correct the curve, and because of persistent paralysis and evidence of a block as shown by lumbar puncture, a laminectomy was performed July 11, 1928, from the twelfth dorsal to third lumbar. The lateral articulations were fused at this time. The neurological condition gradually improved after this. Because of the bad alinement of the trunk it was decided to induce a compensatory curve to the right in the dorsal region and to fuse this area. Following a wedging jacket, a fusion operation was done from the eighth to the twelfth dorsal on April 23, 1929. Another fusion operation was done on May 1, 1929, from the third lumbar to the sacrum, in order to maintain the position of the pelvis in relation to the trunk. Repair of a break in the fusion between the eleventh and twelfth dorsal was made on August 12, 1929. Because the curve was increasing, this area again was exposed on July 30, 1930, and a wide pseudarthrosis was discovered between the eleventh and twelfth dorsal. An attempt was made to repair it. She recovered sufficiently from her paralysis to walk with crutches; discharged from hospital in January, 1931. The curve, originally 50 degrees, measured 73 at that time.

[1]For description of the jacket see Hibbs, Risser, and Ferguson. "Scoliosis treated by the fusion operation: an end-result study of three hundred and sixty cases." J. Bone & Joint Surg., 1931, xiii, 91.

CASE 6. A. H., No. 99,553, a girl 13 years old, came to the clinic of the New York Orthopædic Hospital on April 18, 1927, because of a spinal curvature first noted 4 years previously and which was said to be getting worse. She had had pain for 2 months. Two plaster jackets had been applied without improvement. There was a marked right dorsolumbar, left lumbar curve with rotation. Flexibility of the spine was greatly impaired. X-ray examination showed a left hemivertebra between the third and fourth lumbar, partly fused to the body and arch of the third lumbar. Removal of the hemivertebra was advised and the operation was performed on May 25, 1928, when the body of the hemivertebra and a portion of the body of the third lumbar were removed through a left lumbar incision. The patient was in slight shock but left the operating room in good condition after an infusion of glucose. On June 11, 1928, 17 days after the first operation, the spinous process, lamina, and pedicle of the hemivertebra were removed through a dorsal midline incision. Bleeding was quite profuse when the pedicle was removed but was controlled by packing. The lower part of the lamina of the third lumbar was removed in order to make possible the correction of the curve. One week after operation a wedging jacket was applied, by means of which the curve was overcorrected. The patient was discharged from the hospital in a jacket on July 23, in the hope that the bodies of the third and fourth lumbar would fuse. X-ray examination on September 3 indicated that this had not occurred. On October 1, 1928, a spine fusion of the posterior arches of the first to fourth lumbar was done. On May 10, 1932, she had no pain in her back but did have occasional pain in the scar over the anterior part of the iliac crest. Her spine was straight and fusion of the lumbar region seemed solid. She was doing housework. X-ray examination showed the spine straight and fusion very strong. She had a right curve of 7 degrees, as compared with a left curve of 37 before operation, having been overcorrected.

CASE 7. W. B., No. 89,167, a colored girl, 15 years old, presented herself at the clinic of the New York Orthopædic Hospital on February 5, 1926, with a lateral curvature of the spine, which was noted 8 years before, complaining of pain in her left side. There was a pronounced right lateral curvature in the lower dorsal region, with marked rotation of the vertebræ and compensatory curves above and below. X-ray examination showed that the primary curve extended from the eighth dorsal to the first lumbar and that the angle was 100 degrees. Correction by means of plaster jackets, followed by spine fusion, was advised. The patient did not enter the hospital until November 3, 1927. Traction jackets were applied, and the right dorsal curve was reduced to 81 degrees. It was decided that effective correction could be obtained only by partial removal of a vertebral body. On December 21, 1927, through a dorsal incision to the right of the midline, a part of the lamina and the pedicle of the tenth dorsal vertebra were removed. The right half of the body of the tenth dorsal was excised and the remaining half was divided transversely. There was comparatively little bleeding. The patient ran a high temperature and leucocytosis after operation, and at the end of 8 days pus was evacuated from the wound, the culture from which grew staphylococcus albus. A wedging jacket was applied, by means of which the curve was reduced from 80 to 55 degrees. Through a window in the jacket a spine fusion operation was done from the fifth dorsal to the first lumbar on March 22, 1928. The incision healed by primary union. She was discharged from the hospital, in a jacket, on July 15, 1928, at which time the X-ray film indicated that there was a pseudarthrosis in the fusion area. She was readmitted to the hospital and a repair of the pseudarthrosis, which occurred at the site of removal of the hemivertebra, was done on September 21, 1928. A kyphos had developed at this level. In January, 1930, the curve had increased to 69 degrees. An exploration of the area of fusion was done on January 15, 1930, and it was found to be solid. The second and third lumbar vertebræ were added to the fusion. In November, 1931, the angle of measurement was 79 degrees and the kyphos at the site of vertebral removal was considerable.

CASE 8. Y. N., No. 81,365, a girl, 11½ years old was taken to the New York Orthopædic Hospital on March 23, 1925, because of a curvature of the spine which had been discovered 1 year before. She was a healthy, well developed child with a somewhat severe right dorsal curve with moderate rotation and compensatory curves above and below. X-ray examination showed that the primary curve extended from the sixth dorsal to the second lumbar with an angle of 52 degrees. Correction and fusion were advised but were refused. She returned to the clinic in July, 1927, having had chiropractic treatment and a cast set. The curvature and rotation had increased markedly, the measurement of the curve now being 84 degrees. The family finally consented to operation, and she was admitted to the hospital in December, 1927, at the age of 14 years. By means of a wedging jacket, the right dorsal curve was reduced from 84 to 55 degrees, and on February 3, 1928, through a right dorsal incision, an exposure was made of what was thought to be the tenth dorsal vertebra. About one-third of the body was removed, but the posterior cortex could not be gotten out cleanly and less of the body than was intended was removed because of profuse bleeding from the posterior venous sinus. An infusion of glucose was given as the wound was being closed and this was followed by a blood transfusion. A right pneumothorax was present after the operation, indicating that the pleura had been opened. This gradually absorbed and the patient recovered quite promptly from a severe postoperative reaction. X-ray examination showed that the bone had been removed from the body of the ninth rather than the tenth dorsal, as was thought at operation. A wedging jacket was applied on February 18, by means of which correction was obtained to 64 degrees. Because of the patient's general condition and several pressure sores which developed from the jacket, spine fusion was not done until September 28, 1928, at which time the tenth dorsal to third lumbar were included. On November 2, the fourth to tenth dorsal were added at a second operation. An X-ray was taken in June, 1929, at which time the curve was 64 degrees and the fusion appeared doubtful between the ninth and tenth dorsal. When the jacket was removed in August, 1929, the curve was 70 degrees. Because of continued pain and X-ray evidence of pseudarthrosis, an operation was done on December 11, 1931, disclosing a wide area of non-union between the ninth and tenth dorsal. This was repaired and the patient was discharged from the hospital, in a jacket, on February 25, 1932. In addition to a curve of 76 degrees she had a moderate kyphosis at the ninth dorsal.

CASE 9. B. V. H., No. 63,375, had poliomyelitis when a girl 5 years old. A lateral curvature of the spine was first observed when she was 12 years old. She was seen at the clinic of the New York Orthopædic Hospital on May 2, 1923, when she was 17. At that time she had a severe right dorsal, left dorsolumbar curve with exaggerated posterior deformity. The upper curve, extending from the second to the tenth dorsal, measured 45 degrees, and the lower, from the tenth dorsal to the fifth lumbar, measured 43 degrees. These curves were practically unaffected by traction jackets. She was admitted to the hospital, and double subastragalar arthrodeses and double correction of hip flexion deformities were done in November and December, 1923. By means of jackets with lateral hinge and turnbuckle the

Fig. 7.

Fig. 8.

Fig. 9.

Fig. 7. Case 9. Lateral curvature before operation.
Fig. 8. Case 9. After operation. Portions of the first and second lumbar bodies have been removed.

Fig. 9. Case 9. X-ray picture taken 4 years after operation, showing correction of the curve and fusion of the bodies.

lower curve was reduced to 21 degrees. A spine fusion was done from the tenth dorsal to fourth lumbar on July 23, 1924. One year after operation, following an injury from a fall, X-ray examination showed an apparent defect in the fusion between the twelfth dorsal and first lumbar and an increase in the deformity. The spine was explored on March 18, 1926, and a line of fracture through the center of the fusion mass was repaired. In April, 1927, it was noted that her trunk listed to the right and this became more marked, throwing her badly off balance. Because of this it was decided to remove part of two bodies on the convex side of the curve, the only possible way in which the deformity could be corrected and balance restored. On May 7, 1928, through a dorsal incision, a wedge was removed from the fused laminæ, 1 inch wide at its base, on the left side, as well as a wedge-shaped piece of the adjacent parts of the bodies of the first and second lumbar vertebræ, including about half of each body and the intervertebral disc. Nine days after operation a traction jacket, with anteroposterior hinges, was applied, by which the curve gradually was reduced from 30 to 6 degrees. A jacket was worn until June 27, 1929. An X-ray examination in January, 1932, showed solid fusion of the posterior arches from the tenth dorsal to the fourth lumbar and of the remaining portions of the bodies of the first and second lumbar. She had occasional fatigue but no pain. There was a list of the trunk to the right side but her posture had been greatly improved over that before the removal of the body.

Case 10. F. B., No. 16,592, a boy, 3½ years old, had an attack of poliomyelitis in September, 1916. He was seen in the out-patient department of the New York Orthopædic Hospital on November 15, 1916, with involvement of the left lower extremity. It was noted in 1925 that there was a right dorsolumbar scoliosis, which corrected largely with traction. In August, 1926, there was a marked lateral curvature with pronounced rotation. X-ray examination showed that this extended from the seventh dorsal to the third lumbar, with an angle of 64 degrees. Spine fusion, after correction in a jacket, was advised, but the parents refused to have this done. In November, 1927, the curve had increased markedly, measuring 80 degrees. He was then 16 years old. He was admitted to the hospital in March, 1928. By means of a hinged jacket the curve was reduced to 31 degrees. On May 10, 1928, an operation for partial removal of the bodies of the twelfth dorsal and first lumbar was performed, through a window in the jacket, by a dorsal approach. Because of profuse hæmorrhage the operation was followed by a blood transfusion. Sixteen days later a new traction jacket with anteroposterior hinges was applied. On August 30, the curve measured 18 degrees. It was decided to fuse the spine from the fourth dorsal to the fourth lumbar in several stages. The first included the ninth dorsal to the first lumbar and was done on September 4, 1928. Three weeks later a fusion operation was done from the fourth to the ninth dorsal, and on November 9 the first to fourth lumbar were added. At this time a considerable kyphos was present at the dorsolumbar junction. He was discharged from the hospital on March 17, 1929. The jacket was removed in October, 1929. Following this he had pain in the dorsolumbar region and the X-ray picture indicated that fusion was not solid at the first lumbar. The curve measured 23 degrees in April, 1931. There was tenderness at the twelfth dorsal and a marked kyphos. Repair of the pseudarthrosis was advised, but the patient refused.

CONCLUSIONS

1. Removal of the body and posterior arch of a hemivertebra is feasible in the lumbar region of the spine and is the only means of correcting a lateral curvature caused by this anomaly. This should be supplemented by a spine fusion operation.

2. The operation should be done in two stages, the first consisting of removal of the vertebral body through a lateral lumbar incision with a retroperitoneal approach. The posterior arch should then be removed subperiosteally through a dorsal midline incision.

3. This operation may be used also in certain selected cases of severe lateral curvature in the lumbar region not caused by hemivertebræ.

4. Removal of a vertebral body in the dorsal region is impracticable because of the difficulty in exposing the body and the danger from hæmorrhage and shock.

THE PERNICIOUS ANÆMIA SYNDROME IN GASTRECTOMIZED PATIENTS[1]

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THE intimate relationship between achylia gastrica and the pernicious anæmia syndrome has been demonstrated by many investigators. As further evidence in support of this relationship, the following case is reported.

CASE REPORT

Mrs. V. M., white, housewife, aged 29 years, was admitted to the medical wards on May 1, 1929, with chief complaint, vomiting. The onset of her illness was in February, 1928, 15 months previously, following a miscarriage. At this time, the patient was troubled with frequent vomiting spells, which later became persistent and associated with cramp-like pains in the abdomen. The attacks of vomiting occurred immediately after meals, and were so severe that the patient was unable to retain even fluid. Constipation was marked, there seldom being more than two movements a week. Other symptoms noted were weakness, dizziness, headaches, and a loss of 30 pounds in weight. The significant facts of the past history were four miscarriages, diminished hearing, and occasional generalized twitching of the muscles.

The physical examination revealed a markedly emaciated adult female; hearing diminished in the left ear; and tenderness in the left upper quadrant of the abdomen associated with spasm. A small firm mass was present in this region; the shape was indefinite and the borders could not be outlined. The tumor became more evident when the patient was rotated on her right side. In addition to these findings, there was a generalized lymphadenopathy.

The laboratory report is as follows: The urine was essentially normal. The stools showed no blood or parasites. The gastric contents on analysis, following histamine injection, contained no free hydrochloric acid but much food material from the previous day. The blood picture was as follows: the red blood cell count was 4.2 million per cubic millimeter; hæmoglobin, 70 per cent (Sahli). The Kahn test on two occasions was 4+. The X-ray studies revealed: (1) chest negative; (2) small vertical type of stomach with marked irregularity of the greater curvature, prepyloric, and stenosis of the antral end. In connection with the history, the appearance was suggestive of hereditary syphilis. Fluoroscopic examination showed the isthmus to be completely shut off.

Course. The patient received a series of arsphenamine treatments with no benefit. Consequently a partial gastrectomy was performed according to the method of Pólya. The pathological report was as follows: "Chronic ulcer with vascular granulation tissue base backed by dense fibrosis. Plasma cell and lymphocyte infiltrations. No neoplasm. Highly suspicious of syphilitic ulcer. No spirochetes found."

The patient made an uneventful recovery and felt comparatively well until June, 1931. Following an appendectomy at this time, she again had frequent vomiting spells, epigastric distress, "heart burn," and a throbbing sensation in the abdomen. Two weeks previous to her second admission, June, 1932, she fainted. The patient also noticed increasing weakness, pallor, poor appetite, and a loss of 10 pounds in weight. Physical examination at this time revealed a pale, emaciated, adult female, with marked tenderness in the abdomen. The Kahn test was again 4+. A

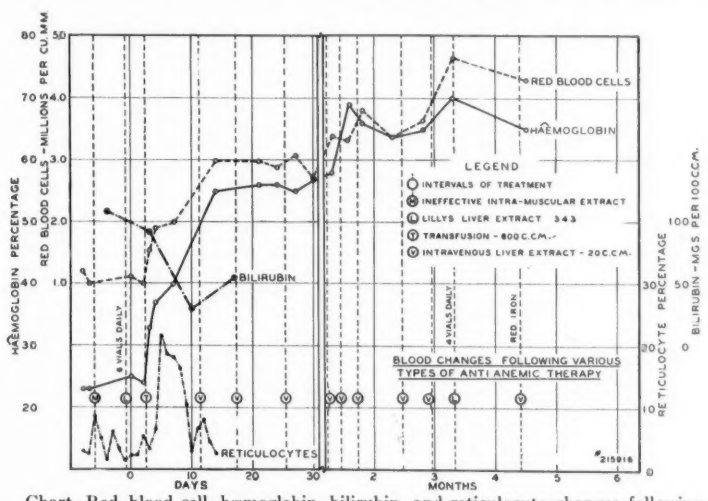

Chart. Red blood cell, hæmoglobin, bilirubin, and reticulocyte changes following various types of anti-anæmic therapy.

[1]From the Thomas Henry Simpson Memorial Institute for Medical Research, University of Michigan, Ann Arbor, Michigan.

TABLE I

Date	Red blood cells. Million per cu.mm.	White blood cells per cu.mm.	Hæmoglobin per cent (Sahli)	Reticulocyte per cent	Treatment	Day of treatment	Measurements of red blood cells on 6–14–32
							Microns 4.5 M—0
6–14–32	1.12	3350	25	2.1	Lilly's No. 343, 6 vials daily	0	
6–15–32				2.2		1	6.0 6
6–16–32	1.01	3900	24	5.4	Transfused	2	6.7 6
6–17–32	1.54	4750	33	3.3		3	7.5 32
6–18–32	1.92	4050	37	6.5		4	8.2 28
6–19–32				21.9		5	9.0 20
6–20–32				18.9		6	9.7 3
6–21–32	1.99	3400	40	18.1		7	10.5 2
6–22–32				16.4		8	12.0 0
6–23–32				10.3		9	13.5 0
6–24–32				3.1		10	
6–25–32				6.9		11	
6–26–32				8.1	Oral liver extract discontinued. Intravenous liver extract	12	
6–27–32				4.5		13	
6–28–32	2.97	4950	55	2.8		14	
7–5–32	2.96	6200	56		7–2–32. Intravenous liver extract	21	
7– 8–32	2.84	5700	56		7–9–32. Intravenous liver extract	24	
7–11–32	3.07	7350	55			27	
7–14–32	2.74	6450	57		7–16–32. Intravenous liver extract	30	
7–23–32	3.39	6450	58		Intravenous liver extract	39	
7–30–32	3.33	8100	69		Intravenous liver extract	46	
8– 6–32	3.78	12250	66		Intravenous liver extract	53	
8–20–32	3.42	8150	64		Intravenous liver extract	67	
9– 3–32	3.65	6850	65		Intravenous liver extract	81	
9– 7–32	4.65	9300	70		Intravenous liver extract	95	
10–22–32	4.30	5700	65		Intravenous liver extract. + Fe	130	

"gastric" analysis with histamine hydrochloride showed no free acid. A blood count and film at this time were typical of pernicious anæmia. The red cell count was 1.03 million per cubic millimeter; the white cell count was 3800 per cubic millimeter; the hæmoglobin was 23 per cent (Sahli); the differential leucocyte count was as follows: polymorphonuclear neutrophils, 59 per cent; eosinophils, 2 per cent; large lymphocytes, 34.5 per cent; small lymphocytes, 3.5 per cent; monocytes, 0.5 per cent; and hemohistocytes 0.5 per cent. The measurement of the red cells showed a marked variation in the size of the cells, with 55 per cent of the cells larger than 7.5 micron. The bilirubin was increased in amount, being 1.17 milligrams per 100 cubic centimeters of blood. A summary of the blood findings is shown in the chart and Table I.

The patient was given a blood transfusion immediately because of her serious condition, and then given liver extract therapy, orally and parenterally, with a response characteristic of pernicious anæmia. For 12 days, 6 vials of liver extract (Lilly No. 343) were administered daily, and following this, intravenous treatment was instituted at weekly, biweekly, and monthly intervals. The patient has shown steady improvement, and, when last observed, her blood picture was as follows: red blood cell count, 4.30 million per cubic millimeter; white blood cell count, 5100 per cubic millimeter; and hæmoglobin, 65 per cent (Sahli).

In Table II is listed a summary of the findings from the cases reported in the literature concerning the association of gastrectomy and a pernicious anæmia syndrome. In all, there were 13 cases, including ours. Operation was performed on 9 occasions for ulcer of the stomach; on 0 occasions for malignancy; and on 4 occasions for questionable but highly probable syphilis of the stomach. The type of operation varied. Total and partial resections were common and in 4 instances gastrojejunostomies were performed. Anæmia developed in from 5 months to 15 years following the operation. Poole and Foster reported a case with a red count of 550,000 per

TABLE II

No.	Author	Age	Lesion	Operation	Interval	Red blood cell count*	Hæmoglobin per cent	Treatment**	Cord changes
1	Moynihan	43	Malignancy	Gastrectomy	3 yrs. 8 mo.	Severe	anæmia	–	–
2	Hartman	58	Malignancy	Gastrectomy	3 yrs.	1.40	48	–	–
3	Ellis	56	Malignancy	Gastrectomy	15 yrs.	2.00	40	–	+
4	Breitenbach	57	Malignancy	Gastrectomy	6½ yrs.	1.80	39	–	–
5	Hochrein	55	Malignancy	Gastrectomy	8 yrs.	1.50	40	+	–
6	Morawitz	56	Malignancy	Gastrectomy	8 yrs.	1.56	54	+	–
7	Morawitz	69	Malignancy	Gastrectomy	6 yrs.	.80	19	+	–
8	Ungley	41	Malignancy	Gastrectomy	5 mos.	3.81	95	+	–
9	Berger	69	Malignancy	Gastrectomy	6 yrs.	.95	20	+	+
10	Cohn	–	Malignancy	Gastrectomy	–	–	–	–	–
11	Campbell and Conybeare	–	Ulcer	Gastrojejunostomy	8 yrs.	–	–	–	–
12	Glanvill and Hurst	34	Ulcer	Gastrojejunostomy	3 yrs.	1.68	44	+	+
13	Fairley and Kilner	42	Ulcer	Gastrojejunostomy	3 yrs.	3.60	65	+	–
14	Willcox	52	Ulcer	Gastro-enterostomy	2 yrs.	–	–	–	–
15	Delore	43	Ulcer	Gastropylorectomy	5 yrs.	.68	17	–	+
16	Dennig	41	Ulcer	Gastrectomy	7 yrs.	1.80	40	+	+
17	Hochrein	56	Ulcer	Gastrectomy	9 yrs.	Severe	anæmia	+	–
18	Scheidel	50	Ulcer	Gastrectomy	6 yrs.	.63	22	+	–
19	Hangarter	–	Ulcer	Gastrectomy	2 yrs.	2.39	50	+	–
20	Poole and Foster	39	Syphilis	Gastrectomy	3 yrs.	.50	20	+	–
21	Rowlands and Simpson	38	Syphilis	Gastrectomy	6 yrs.	2.10	40	+	+
22	Crohn	–	Syphilis	Gastrectomy	–	–	–	–	?
23	Goldhamer	29	Syphilis	Gastrectomy	3 yrs.	1.0	25	+	–

*The red blood cell count in millions per cu. mm. before treatment was instituted.
**Various types of liver, liver extract (orally or parenterally), and Ventriculin.

cubic millimeter; Scheidel and Delore reported two other cases with counts of 600,000 cells per cubic millimeter. Six of the cases had cord changes. The other symptoms most commonly noted were those associated with anæmia—weakness, tiredness, ease of fatigue, pallor, dyspnœa, and palpitation. Of the 14 cases treated with liver therapy, orally or parenterally, a response was obtained exactly like that of any uncomplicated case of pernicious anæmia.

According to Poole and Foster, anæmia occurs in all gastrectomized patients if they survive long enough. Rowlands and Simpson concluded that the occurrence of primary anæmia after gastric operations was not a mere coincidence. Since only one case has been reported with an anæmia occurring within a period of 24 months after operation, and several cases of gastrectomy have been reported with no anæmia occurring within this interval, the time element must be a most important factor in the development of the anæmia. A patient may then have the predisposing factors of pernicious anæmia for at least 6 to 24 months before any of its characteristics manifest themselves clinically.

It is now necessary to answer the question of whether the anæmia associated with gastrectomy is the result of the operation, or whether it is an incidental complication. Carcinoma of the stomach as well as syphilis has been reported to cause blood pictures similar to that of pernicious anæmia. In the previous reported cases as well as in our own, either no anæmia was noted previous to the operation, or it was of the secondary type. Since our patient had syphilis for a long period of time presumably sufficient to produce an anæmia, and yet had none, it is reasonable to conclude that the present anæmia is the direct result of the gastric resection. However, further proof is necessary before a definite statement can be made.

The development of pernicious anæmia following gastrectomy appears to be further evi-

dence in support of the theory that some function of the stomach is a factor in the prevention of this malady. Any agent which would destroy this function, whether it be disease or the removal by operation, would give the same resulting blood picture, that of pernicious anæmia.

From a study of the blood findings, it is apparent that with liver therapy the red cells have returned to a normal level and have been properly maintained. However, the hæmoglobin still remained deficient. This is suggestive of the fact that there is also a disturbance of the iron metabolism as a result of the gastrectomy. Secondary anæmia has been produced experimentally on several occasions by Ivy in gastrectomized dogs. Clinically, the relationship between iron deficiency and achlorhydria has often been described in the syndrome "chronic microcytic anæmia" or "simple achlorhydric anæmia." The results of this case, as well as that of Morawitz's case, would seem to be in accordance with the experimental and clinical findings discussed.

SUMMARY AND CONCLUSIONS

1. A patient with chronic gastritis, probably syphilitic, who developed symptoms indistinguishable from pernicious anæmia 2 years following gastrectomy, is described.

2. Regardless of the initial lesion in the stomach, or the type of resection performed, the blood picture and symptomatology of pernicious anæmia may develop after a latent period of 5 months to 15 years following the operation.

3. The anæmia resulting from gastrectomy responds satisfactorily to liver therapy.

4. A possible relationship between iron metabolism and normal gastric function is suggested.

Since this paper has been written, Rowlands and Simpson, and Ungley, have published similar data. Their additional cases have been incorporated in the statistics of this paper.

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PHENYL MERCURY NITRATE

Its Clinical Uses in Gynecology, A Preliminary Report

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RECENTLY, Weed and Ecker reported on the use of phenyl mercury nitrate and related compounds as disinfecting and antiseptic agents.[1] They found that phenyl mercury nitrate combines with a very high bactericidal potency a relatively very low local and systemic toxicity for animals and for man. Its effectiveness is not impaired by the presence of tissues or tissue juices; it is but very slightly irritant on local application to wounds and most mucous membranes; it has no odor or color and does not stain; it does not corrode surgical instruments.

Accordingly, at the suggestion of Professor E. E. Ecker, we undertook a study of this compound, limited to the treatment of infections in the vagina and cervix. The phenyl mercury nitrate for this investigation was supplied by Drs. Weed and Ecker.

This series includes 70 consecutive cases of infections in these areas which were treated as indicated below.

As there was no previous clinical work with this antiseptic agent reported, it was thought advisable to begin the treatment with very high dilutions, concentrating the product as conditions warranted.

In this report it is to be noted that no attempt was made to select certain types of cases. All consecutive patients seen by the author showing a vaginal or cervical discharge were treated with phenyl mercury nitrate.

PROCEDURE

The following is an outline of the standard procedure used: Upon separation of the labia, the urethral orifice was exposed and a smear of the urethral secretion (if present) was taken routinely. The vagina was then opened with the usual bivalve speculum and the cervix was exposed. A cervical smear was then made. Subsequently the entire vagina was cleansed with dry absorbent cotton balls and the cervical canal wiped clean with a dry cotton applicator. Phenyl mercury nitrate was then instilled into the cervical canal by means of a cotton applicator and it was held *in situ* while a cotton ball saturated with the medication was used to swab out the vagina thoroughly. The applicator was then removed and a tampon, the distal end of which was saturated with phenyl mercury nitrate, was inserted. The patient was instructed to remove the tampon after 24 hours and then to begin the daily douche.

Dilutions of 1:25,000 and 1:12,500 were used at first, but it was soon evident that lower dilutions could be used with safety. Continued experimentation afforded sufficient evidence that the optimum results were obtained by using a 1:1,250 solution, which, according to Weed and Ecker, is the saturation point for phenyl mercury nitrate in water.

Originally the patients were directed to use a daily douche in connection with this treatment consisting of a teaspoonful of sodium bicarbonate to a quart of moderately warm water. Instructions were issued to each patient to avoid the use of salt in the douche in view of the resultant relatively insoluble phenyl mercury chloride which would be formed from the nitrate in the presence of excess chloride. After some experimentation, it was found that 1 gram of phenyl mercury nitrate could be dissolved in 100 cubic centimeters of pure glycerine and so dispensed by the outpatient pharmacy. One dram of this solution thoroughly mixed with a quart of water produced about a 1:25,000 dilution which could be used as a douche.

The first 3 cases treated received phenyl mercury nitrate in a dilution of 1:25,000 and were instructed to use the alkaline douche. In the succeeding 9 cases the patients received the medication in a dilution of 1:12,500 with the aforementioned bicarbonate of soda douche. In the 58 remaining cases a dilution of 1:1,250 was used. In 5 of these, during one phase of the treatment, phenyl mercury nitrate in a dilution of 1:750 in 10 per cent alcohol was used in an attempt to determine the results with a more concentrated solution. The use of the 1 per cent glycerine solution diluted to 1:25,000 in water as a douche was inaugurated sufficiently early in the course of the study to permit its use in 48 patients in conjunction with the 1:1,250 instillation.

DIAGNOSES

In 13 cases the condition was clinically found to be acute gonorrhœal endocervicitis and the diag-

[1] J. Infect. Dis., 1931, xlix, 440-449; ibid., 1932, li, 309-314.

nosis was substantiated by laboratory findings. In 8 cases the diagnosis was acute gonorrhœal endocervicitis clinically, but it was not substantiated by the laboratory. Three patients with vaginitis were treated, one due to the persistent use of hot douches, the second to the irritation produced by a pessary with subsequent secondary infection, and the third, that of a child 3 years old with an acute gonorrhœa. One patient with a postoperative rectovaginal fistula responded to treatment with sufficiently interesting results to warrant devoting a paragraph to an outline of the history which will be found below. Two patients with Trichomonas vaginalis were treated with phenyl mercury nitrate among other medicaments. One patient with an incision and drainage of a Bartholin abscess with the instillation of phenyl mercury nitrate is to be noted.

The 42 remaining cases consisted chiefly of various forms of endocervicitis associated with a host of other conditions referable to the pelvis, ranging from chronic cervicitis as the result of old lacerations to chronic ulcerative endometritis, the result of radium implantation.

Thirty-six of the 70 cases received some form of treatment prior to beginning the use of phenyl mercury nitrate. It is not in the province of this paper to go into any detailed comparison of phenyl mercury nitrate with other antiseptics. It is essential, however, that the reader, in evaluating these results, should know in which cases other forms of treatment had been used. In 34, phenyl mercury nitrate in one or another of the dilutions was the initial and only treatment used.

In all cases smears were taken and a notation made on the dispensary chart of the clinical picture with particular reference to the color, consistency, amount, and odor of the vaginal discharge. Subsequent records embodying these details were made at the time of each visit.

SPECIFIC ENDOCERVICITIS

Of 21 cases of definite clinical gonorrhœal endocervicitis, a large number of the patients had been resistant to various other forms of treatment. Subsequently these patients were treated with phenyl mercury nitrate according to the method outlined. Without one exception each patient reporting back to the dispensary within 3 to 7 days indicated that her discharge had lessened or disappeared entirely and that she felt very much better. Examination, however, showed that while these patients were subjectively improved and no discharge was in evidence to them, objectively the improvement at the second visit was not so pronounced as they indicated.

In other words, one application of phenyl mercury nitrate could not and did not completely relieve the patient. Upon inspection, however, it was evident that the amount of the discharge was distinctly reduced. It is safe to say that the reduction in quantity amounted to 50 per cent or more. It was the rule to instruct all our patients not to douche on the day they returned for treatment. What to us was of prime importance was the fact that the color and consistency of the discharge had changed from a thick greenish-yellow, foul-smelling leucorrhœa to a milk white, odorless, and comparatively thin secretion. This change in the discharge was seen in our very early cases, and it became very much more pronounced in those patients who had the benefit of phenyl mercury nitrate as a douche.

These cases of gonorrhœal endocervicitis received treatment with phenyl mercury nitrate for periods varying from 2 to 12 weeks and were subsequently cared for with whatever adjuvant treatment the condition then indicated. In other words, the phenyl mercury nitrate was used to clean up the secondary infection, to eradicate the primary infection, and then to maintain the areas clinically free from infection while other treatment was used to heal associated conditions, e.g., silver nitrate or the actual cautery for cervical erosions.

A typical case as an example of this group is given herewith:

M. M., age 23 years, was referred to the gynecological service on February 10, 1932, from the postnatal division of the obstetrical service, because of a persistent discharge with positive smears for gonorrhœa. Upon examination she showed a typical acute inflammatory process involving the cervix with a surrounding vaginitis. The discharge was yellow green in color and comparatively thick. The fundus was still slightly enlarged, undergoing involution. The adnexa were negative. Smears taken on her first visit showed gram negative intracellular diplococci. Treatment was instituted at once consisting of phenyl mercury nitrate instillations three times weekly and daily douches. On the third visit, 6 days later, the patient volunteered the information that her discharge had disappeared and that she felt "wonderful." Examination at this time revealed a very definite lessening of the inflammatory process, as well as a marked decrease in the amount of the leucorrhœa, with clinical evidence of the change in the character of the infection. Treatment was continued without interruption for another 11 days with excellent results. Gradually the instillations were given at longer intervals, and finally discontinued, when, on March 25, 6 weeks after her first visit to the dispensary, the discharge had completely disappeared. Subsequently silver nitrate was used to treat an associated cervical erosion, and the patient was discharged as cured on April 29, 1932, with repeated negative smears and negative pelvic findings.

VAGINITIS

Similar good results were obtained in the treatment of the 2 cases diagnosed as vaginitis, one

from the irritation due to a pessary with secondary infection, and the other to the persistent use of hot douches. In these cases rapid early improvement was noted and the phenyl mercury nitrate douches were continued until complete eradication of the secondary infection followed. The third case of vaginitis in our series was one of gonorrhœal origin in a child of 3 years. She received only two treatments at our hands with beneficial results. Continued care of this case was prevented by the transfer of the patient to another dispensary.

BARTHOLIN ABSCESS

The results obtained in the 1 case of a Bartholin abscess of right labium are as follows:

This patient had the abscess incised, drained, and packed with iodoform gauze on December 5, 1931, in the dispensary. The following day it was noted that the packing apparently had been lost and the abscess was still draining. About 5 cubic centimeters of the 1:1,250 solution of phenyl mercury nitrate was instilled directly into the cavity by means of a blunt syringe. Because of an associated endocervicitis the patient was given the phenyl mercury nitrate douche for daily use. Two days later the cavity appeared absolutely clean. The combined treatment was repeated and continued until December 30, 1931, when the wound was completely healed and the endocervicitis had cleared up. The patient was discharged as well.

RECTOVAGINAL FISTULA

The following is the case of postoperative rectovaginal fistula mentioned above in which phenyl mercury nitrate was used with beneficial results:

This patient came to our attention in the dispensary on February 17, 1932, after having had a bilateral vaginal incision and drainage for tubo-ovarian masses at two different operations 3 weeks apart, and, 1 month later, a supracervical hysterectomy. Shortly after the last operation a rectovaginal fistula was discovered and she was referred back to the dispensary for treatment. After many futile attempts were made to keep the area clean with various antiseptics at our command, we decided to employ phenyl mercury nitrate. We felt that herein was an ideal case in which to try an antiseptic which might keep the area around the fistula sufficiently clean to allow it to heal. With this in mind we started 1:1,250 phenyl mercury nitrate instillations three times weekly into the cervix and the vagina, and advised the twice daily use of the phenyl mercury nitrate douche. Examination on her first visit revealed a foul greenish-yellow discharge mixed with fæces coming from a fistulous opening about 3/8 inch in diameter just below the cervix. One week after the first treatment it was noted that the discharge was white in color for the first time. Some three weeks later the fistulous opening was found to be thoroughly clean with no evidence of any secondary infection. At this time the instillation of phenyl mercury nitrate was discontinued and the edges of the fistula were touched up with silver nitrate to stimulate epithelial growth. Meanwhile the douches were continued once instead of twice daily. Repeated examinations thereafter revealed a gradual closing of the fistula, while the discharge had completely stopped, and on March 30, 1932, the fistula was found to be definitely closed. At subsequent visits the slight cervical erosions present were treated with silver nitrate and the patient was discharged as well on June 16.

TRICHOMONAS VAGINALIS

Among the forms of protozoan life resistant to ordinary antiseptics is the organism known as Trichomonas vaginalis. In our particular series we had two cases of vaginitis due to this organism which had received courses of treatment with every antiseptic available to us, with very poor results. To determine the efficacy of phenyl mercury nitrate, these 2 patients were treated with this medication in both its forms, as outlined herein. Our results were uniformly poor, establishing to our satisfaction that phenyl mercury nitrate in the saturated solution with the use of the 1:25,000 douche has no inhibitory effect, *in vivo*, on the growth of the protozoan, Trichomonas vaginalis.

NON-SPECIFIC ENDOCERVICITIS

The final series of cases consisted of a group of 42 patients having endocervicitis of a non-specific origin with or without other associated pelvic conditions. These patients received phenyl mercury nitrate in various dilutions by the method described. Twenty-six of these, after receiving the instillations, were instructed to use the bicarbonate of soda douche. The 16 remaining used the phenyl mercury nitrate 1 per cent glycerine douche.

The reports from this group of patients returning on the second visit varied from that of a change in color and decrease in amount of the discharge to those reporting its complete absence. The chief factor of interest in this group is the fact that phenyl mercury nitrate reduced the usual secondary infection to be found in the vagina and cervix in this class of patients permitting the use of whatever adjuvant treatment the associated conditions warranted. A fairly large number of these patients were referred to the House service for various operations limited to the pelvis. Others received the additional necessary treatment in the dispensary.

We were struck by the clinical fact that phenyl mercury nitrate could within 24 to 48 hours change the character of the infection. Whereas we were unable in every case in this group to clear up its source, we were fortunate in being able to reduce the discharge. Many of these patients having such associated conditions as salpingitis in various stages derived great benefit from the use of the hot daily douche with phenyl mercury nitrate.

HIGHER CONCENTRATIONS

In an attempt to obtain a more concentrated solution of phenyl mercury nitrate it was dissolved in 10 per cent alcohol, producing a 1:750 solution. Five of our patients having an endocervicitis of a non-specific origin, in the earlier part of the investigation, were treated with this increased concentration. Each patient after two instillations and tamponing showed a gray film on the cervix only, which, when rubbed, came away. This we took to be a chemical burn produced by the phenyl mercury nitrate. After 1 week's rest, during which time these patients received one instillation of dry boric powder, the burns healed. This concentration of phenyl mercury nitrate was then discontinued and the saturated aqueous solution (1:1,250) substituted, with the usual favorable response given to this medication in this dilution.

SUMMARY

A series of 70 cases of infections of the vagina and cervix treated with phenyl mercury nitrate is reported.

Favorable response to the use of this antiseptic was invariable in all the conditions encountered, both specific and non-specific, except when the Trichomonas vaginalis was the inciting agent.

The character of the infection can be changed, often with but a single application of this antiseptic, with resultant rapid amelioration of the condition.

The results of the use of phenyl mercury nitrate in this series ranged from rapid and complete clinical recovery to improvement sufficiently marked that adjuvant treatment could be successfully applied.

In effective concentrations, both as local application (1:1,250) and as douche (1:25,000), phenyl mercury nitrate is non-toxic; it is practically non-irritant to the vaginal mucous membrane; it is effective in the presence of tissues; it is odorless, colorless, and does not stain; it does not corrode surgical instruments; its solutions show no apparent deterioration on standing.

Phenyl mercury nitrate, being a highly effective bactericidal agent of the characteristics indicated, appears to offer encouraging possibilities in gynecology.

ADDITIONAL CASES

Since this paper was submitted for publication a series of 30 additional cases, treated with phenyl mercury nitrate, has been completed. This work was done in a semi-public institution for indigent girls, in Cleveland. All the inmates were subjected to routine vaginal examinations; smears for gonococci were made and blood Wassermann reactions were determined as part of the venereal disease control program of the Cleveland Health Department. The author was afforded the privilege of doing this work for a 4 month period beginning October 18, 1932. As these patients were institutionalized throughout the period of treatment, these results afforded an excellent check on the series of dispensary patients previously studied.

The individuals ranged in age from 13 to 27 years. All those selected for treatment showed some form of endocervicitis, 19 being specific, clinically and/or microscopically, and eleven non-specific. Of the 30 cases, 17 had had some form of treatment previously, for periods of up to a year or more. The 13 remaining were new inmates examined for the first time.

The method used was identical with that outlined above. These patients were treated twice a week by the author and were instructed to use the phenyl mercury nitrate douche at stated intervals, usually daily. The results were uniformly good, with but a single exception; in the latter case an endocervicitis of specific origin was cleared up, but because of a subacute unilateral salpingitis, the discharge persisted.

We were gratified to note the ease with which the treatment was carried out, causing little disturbance in daily routine.

In order to learn whether the repeated daily use of phenyl mercury nitrate resulted in the absorption of significant quantities of mercury, which among other effects, might prove deleterious to the kidneys, Miss N. E. Schreiber of the Department of Pharmacology, Western Reserve University, determined for us, by a highly sensitive microchemical method, the mercury content of 24 hour urine specimens of three individual cases treated at this institution. These patients were chosen for this purpose because they had used the douche daily for the longest period of time, namely 6 weeks. The urine specimens were collected 24 hours following cessation of all treatment. Miss Schreiber reported as follows:

> Chemical analysis of three specimens of urine received December 1, 1932, for the presence of mercury, shows as follows:

	Volume	Total metallic mercury
M. H.	1200 c.cm.	Trace—about 0.008 mgm.
J. J.	1860 c.cm.	0.022 mgm.
H. W.	1690 c.cm.	0.104 mgm.

While this data is not conclusive, in that no information is available as to the quantity retained in the body or as to the fæcal excretion, nevertheless, judging from the very extensive studies of Schreiber, Sollmann, Cole and associates[1] on the clinical excretion of mercury, the figures given here may be considered to represent an entirely negligible output. Where significant quantities of ionized mercury are absorbed, as in luetic therapy, the urinary excretion of the metal is very much greater than that reported above.

[1]Sollmann, T. Mellon Lecture 1930, Manual of Pharmacology, 4th ed. 1932.

EDITORIALS

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AUGUST, 1933

ACID OR ALKALI IN THE TREATMENT OF URINARY TRACT INFECTIONS

FOR many years it has been believed that changes produced in the reaction of the urine were an important factor in the treatment of urinary infections. On the other hand, opinion has periodically and more or less constantly vibrated between the acid and the alkaline extremes, suggesting no very fundamental convictions in regard to this influence.

For a long time the school of thought which advocated switching the reaction of the urine from acid to alkaline chiefly in infection due to the colon bacillus group and particularly in pediatric practice, held sway. This was based upon the view that an organism which had become accustomed to growth in an acid medium would grow less satisfactorily in an alkaline one. While this was bacteriologically true, it left out of account the ability of the organism rapidly to adapt itself to the changed reaction, and probably neither the degree of acidity nor the degree of alkalinity ever seriously affected the vitality of the organism.

Within the last two years, work by Helmholz and also by Clark has, I think, put this question on a secure basis and demonstrated beyond peradventure that acidification pushed to high limits is a very important, and perhaps the most important single factor in the treatment of these conditions. Helmholz worked with children, Clark with adults. Their results agree in principle and differ in detail. The most important of their contributions was the clear demonstration that acidity of the urine produced by a ketogenic diet will sterilize the urine more certainly and more rapidly than when produced by drugs. In children the sterilization produced by ketosis can occasionally be arrived at within forty-eight hours. In adults the time required is longer and the result less spectacular. In children it has been possible to sterilize the urine even in the presence of incomplete drainage of some portions of the urinary tract, a feat which, as far as I know, has never been heretofore achieved. In adults the combination of ketosis with drugs tending to produce acidity, particularly ammonium chloride and ammonium nitrate, is commonly necessary. In children such a combination is less often required. The important thing is that acidity of high grade should be certainly, and if possible rapidly produced. This degree of acidification can be satisfactorily determined only through the estimation of the hydrogen-ion concentration which should be reduced to at least 5.5 and preferably to 5.

The question of whether it is necessary to combine with this treatment the use of urinary antiseptics such as methenamine must be left

for the future to decide. Certain it is that this group of formaldehyde containing drugs is worthless except in a strongly acid urine. It may possibly prove to be true that acidification alone if skillfully handled may have sufficient power without the assistance of formaldehyde.

Old notions die hard, and it is still widely believed that an acid urine in and of itself produces irritation. Acting upon this assumption alkalies are frequently given to relieve pain. The work mentioned, and particularly that of Clark, has convinced me that acidity in and of itself is probably incapable of producing symptoms, while on the other hand, many a patient with tremendous frequency and a neutral urine has been promptly relieved by making the urine sufficiently acid. I have grave doubts whether there is any indication for giving an alkali for the relief of pain, and it is certain that such administration will seriously handicap the control of infection. It is my best guess that the day of the alkali in the treatment of urinary tract infection is over.

In order to get results a definite plan must be followed. The organism causing the infection must be known from the start, the hydrogen-ion concentration of the urine when the patient comes under observation must be recorded, diet and drugs must be used to the extent necessary to reduce the hydrogen-ion concentration to the neighborhood of 5, and this level of acidity must be maintained if possible until the urine is known to be sterile. Many a clear urine contains the germs of disaster. Nothing short of sterilization can be accepted as a criterion of cure. Most failures are due to three causes: failure to identify the organism causing the condition, failure to produce a sufficient degree of acidity, and finally failure to sterilize the urine before stopping treatment. HUGH CABOT.

SEPSIS, ANTISEPSIS, AND ASEPSIS

THE outbreak of the World War found the medical profession entirely unprepared to cope with the gigantic problems confronting it. The unprecedented number of wounds, as well as the severity of the trauma inflicted by high explosive shells, presented a problem which was theretofore unknown.

Practically all wounds received during 1914–1915 were infected, i.e., pus producing microorganisms, also tetanus and Bacillus welchii were usually present. The devitalization of tissue involving the shell tract was allowed to remain without surgical excision. The antiseptics commonly used such as iodine, bichloride of mercury, phenol, etc., failed to prevent infection or cure or arrest it once it became established. It was not until after thorough excision of devitalized tissues, followed by chemical sterilization with sodium hypochlorite, 0.5 per cent solution as advocated by Carrel, that it was possible to do a secondary suture with any degree of success. Primary and secondary sutures were practically unknown during the early years of the War, most all wounds healing by granulation.

Conditions were chaotic in so far as methods of treatment were concerned. There were two schools of thought which, more or less, bitterly opposed the other; one school advocated the use of salines to the exclusion of antiseptics and the other advocated antiseptics.

The treatment of wounds, compound fractures, osteomyelitis, etc., requires sound surgical procedure and judgment. A scrupulous asepsis followed by the intelligent use of a physiological solution of sodium hypochlorite, 0.5 per cent in combination with a hypertonic saline solution, is the best known treatment to prevent infection and to abort it when

once present. These conclusions were personally confirmed to the author by the Surgeon Generals of the American and British armies and by the Consulting Surgeons of the French and Italian armies.

The clinical evidence is so conclusive that we are at a loss to account for the general lack of interest and failure to adopt a method which has been proved by laboratory and clinical experience to be superior to other forms of treatment.

Foster has reported treating by débridement followed by the Carrel method, 304 consecutive cases of compound fractures of the long bones over a period of 15 years. In this series, there were 2 deaths (1 at the end of 4 weeks and the other at the end of 6 months); there was one amputation but not one infection.

Barone, in 77 cases (septic operations and puerperal sepsis) in which treatment was instituted within the first 72 hours, reports a mortality of 10 per cent. In this series, with few exceptions, the infection was due to hæmolytic streptococcus.

Despite such convincing evidence as to the value of a physiological antiseptic, which is non-irritating and non-caustic, we are confronted by those who advocate the use of maggots and vaseline gauze in wounds, compound fractures, and osteomyelitis. Needless complications, including amputations and deaths, result from such ill advised procedures. Certain advocates of these methods make an appeal from the standpoint of results (?), maintaining that the laboratory and scientific viewpoint based upon laboratory findings, should be disregarded. However, results based upon personal opinions without necessary controls or comparisons, are usually very misleading and incorrect. Not infrequently, patients recover in spite of the treatment rendered.

The pages of medical literature reek with unscientific practices and useless antiseptics. There is no antiseptic known which can be used to the exclusion of sound surgical principles and practices. One wonders how long sepsis following the use of worthless antiseptics, will be condoned when methods are available to prevent it. The surgeon can no longer "alibi" himself by charging the omission and commission against the operating room nurse, the innocent catgut, etc.

Notwithstanding the extravagant claims made for many popular methods, the proved teachings of Pasteur and Lister do and will prevail. WM. O'NEILL SHERMAN.

MEMOIRS

JOHN CHALMERS DA COSTA

1863–1933

J. CHALMERS DA COSTA, truly a Philadelphian, the son of George T. Da Costa, a litterateur and bibliophile, was born on November 15, 1863, in Washington, where his parents were temporarily located. His mother was Margaretta Beasley from Beasley's Point, New Jersey. He received his early education at the Friends' Central School, and was graduated from the Towne Scientific School of the University of Pennsylvania in 1882. He was graduated from Jefferson Medical College in 1885, his preceptor being his uncle, Dr. John C. Da Costa, known familiarly as "Uncle John." J. Chalmers was referred to as "Jack" and his cousin, Dr. John C. Da Costa, Jr., who was of dark complexion as "Black Jack."

Dr. Da Costa served thirteen months as an interne at the Philadelphia General Hospital, after which he was appointed assistant physician to the Insane Department of the same institution, and during this time contributed several papers on insanity.

In 1887, he became one of Chapin's assistants in the Pennsylvania Hospital for the Insane. He engaged in private practice in the same year, and was appointed assistant demonstrator of anatomy at the Jefferson Medical College, and one of the clinical assistants of Dr. Samuel W. Gross. His successive advances at Jefferson were: assistant demonstrator of surgery, demonstrator of surgery, chief of surgical clinic, clinical professor of surgery in 1896, professor of principles of surgery in 1900. Dr. Da Costa was the first incumbent of the Samuel D. Gross chair of surgery which was endowed by Maria Gross Horwitz (daughter of Dr. Gross) in 1910, which he held until his death.

He was attending surgeon to the Jefferson Hospital and long a surgeon to the Philadelphia Hospital; later he served as consulting surgeon to the Philadelphia General Hospital, St. Joseph's Hospital and Miseracordia Hospital, and for many years was surgeon to the Pension Fund of the Philadelphia Fire Department.

He was a member of the American Philosophical Society, American Surgical Association, Society of Clinical Surgery, College of Physicians of Philadelphia, Pathological and Neurological Societies of Philadelphia, the Philadelphia Acad-

J. Chalmers da Costa.

emy of Surgery, associate member Society of Gynecology and Surgery of Bucharest, member of the Société International de Chirurgie, the Historical Society of Pennsylvania, formerly Commander U.S.N.R.F., etc.

He loved to teach and his hearers were impressed with his foundation in anatomy, his knowledge of surgery, his familiarity with history, his frequent quotations from literature, and his inimitable manner in presenting a subject. Jack Da Costa always was at his best before a large audience. Only those who saw him before he became incapacitated in 1922 will remember his characteristic attitude while conducting a diagnostic clinic for the students, with amphitheater filled to capacity, the clinic floor and doorway crowded with visiting physicians, confrères, assistants, and former students. First standing to one side of the "pit" with arm resting on the rail and one foot crossed in front of the other, then walking across the floor with body vibrating and knees bending, he spoke, giving clear, systematic, unmistakable facts which left an indelible impression.

He was the idol of the medical students; their admiration was spontaneous. He appealed to the imagination, aroused enthusiasm, and stimulated effort.

As an author his name will live for centuries. In 1894 the first edition of his *Manual of Modern Surgery* was published; the tenth edition appeared in 1931. The last revision was accomplished with much difficulty while he was ill and in great pain. It is one of the most widely known and extensively used textbooks. In the same year he prepared another book for publication entitled *The Papers and Speeches of John Chalmers Da Costa* dedicated to Dr. Harvey Cushing. Many, but not all of his best speeches are contained in this volume; one of the most notable being "Dickens Doctors" which he read before the Philobiblan Club, Philadelphia, May 28, 1903. It is regrettable that he did not include in this book an oration delivered in 1899 at the semicentennial exercises of the Philadelphia County Medical Society which was one of the most masterful productions of his facile pen. Some of his other writings have been: in collaboration with Dr. Frederick Packard on Keating's *Medical Dictionary;* section on "Methods of Dissection" for Nancrede's *Anatomy;* section on "Epilepsy and Tetanus" in Hare's *American System of Therapeutics;* section on "Diseases of the Testicle," etc., in Keating's *Cyclopedia of Children's Diseases;* edited Zuckerkandl's *Operative Surgery* in 1899; first editor with Dr. Keen of Keen's *Surgery;* editor of Gray's *Anatomy;* the *Blood Alterations of Ether Anesthesia*, in 1905; articles upon compound fractures of the skull, amputation of hip joint, sarcoma of tonsil; trephining of skull, blood changes in ether, surgery of insanity, surgery of epilepsy, etc., and various literary topics.

His surgical teaching has permeated every civilized portion of the earth. He taught medical students from most countries, and visiting physicians and surgeons seldom missed an opportunity to attend his clinics. Rarely did he conduct a clinic which was not attended by some notable surgeon.

Dr. Da Costa received his early surgical training under Dr. Samuel W. Gross. Before he became a medical student he used to attend the Saturday clinics at Blockley conducted by Dr. Gross. Of him he said, "He made a most forcible impression upon my mind. His positive character, his clean-cut sentences, his readiness to accept responsibility, his scorn of clap-trap and hatred of boasting, his diagnostic skill and operative ability, all captivated my youthful imagination." The same may well be said of Dr. Da Costa.

He was intimately associated with Dr. W. W. Keen, whose influence and daily contact did much to mold his surgical career. He succeeded Dr. Keen upon his retirement in 1905 as professor of surgery in the Jefferson Medical College. Later his colleagues were Dr. John H. Gibbon and Dr. Francis T. Stewart, deceased, indeed a strong surgical team.

Dr. Da Costa was married to Miss Mary Roberts Brick in 1894, member of a prominent Philadelphia family, who survives him. There were no children.

He became ill with a form of arthritis more than a decade ago. Because of the painful and deforming arthritis, for approximately ten years he conducted his lectures and diagnostic clinics sitting in a wheel chair. He retained his keen mental process until a few days before his death, May 16, 1933.

In the passing of Dr. Da Costa the faculty of Jefferson has lost one of its ablest members; the profession has lost a great teacher, a resourceful surgeon, a distinguished author; Philadelphia has lost a valuable citizen and a man of forceful character.

EDWARD J. KLOPP.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

A LARGE, well printed, thoroughly illustrated, completely referenced treatise[1] on thyroid disease is presented by Joll in *Diseases of the Thyroid Gland*. The first half of the volume is devoted to the pathological anatomy and pathogenesis of goiter and thyroid dysfunction. The next quarter is given over to a discussion of thyrotoxicosis. In the last quarter, operative technique, anæsthesia, postoperative care, complications, and results are clearly presented. The work is to be recommended for the detailed presentation of anatomy and pathology, which will be useful for surgical study or for experimental approach. The discussion of thyrotoxicosis is in agreement with the best American opinion. The surgical technique is presented in useful form. The illustrations are excellent, they number 283, and there are 24 colored plates in addition. This volume of 682 pages fills the place of an authoritative reference work in one volume. It should be of great practical value.

PAUL STARR.

THE material in Kaiser's book[2] on tonsils has been gathered from a careful survey of 4,400 school children and covers a period of 10 years. Although operation was recommended in all cases, only 50 per cent consented, the remaining half being used as controls. Three years after operation a survey was made and again 10 years after operation, and a careful check was made to determine the incidence of the more common complaints of childhood. Statistics gathered bring to light many interesting facts. Apparent benefits during the first few years after operation are not so evident over a 10 year period. This is particularly true of acute head colds and otitis media where the incidence is about the same in tonsillectomized and non-tonsillectomized children after a 10 year interval. Certain conditions are decidedly benefited by the removal of tonsils and adenoids such as acute sore throats and cervical adenitis. On the contrary, infections in the respiratory tract such as laryngitis, bronchitis, and pneumonia seem to be adversely affected.

The book contains 300 pages and 27 chapters. Besides the statistical data there is much concerning the anatomy and physiology and bacteriology of the tonsils. The book offers a ready reference for pediatricians and otolaryngologists and serves to clarify and remove uncertainties regarding the operation for tonsils and adenoids in childhood.

J. F. DELPH.

THE subject of electrosurgery is presented by Kelly in a manual of 305 pages with 382 illustrations.[3] The various operations with electrodesiccation, electrocoagulation, and "acusection," or cutting, are described, as are the preparation of the patient, the anæsthesia, and the postoperative care. The book is largely a regional operative surgery invaluable for any surgeon interested in perfecting himself in electrosurgical technique. The various types of current and their specific effects are discussed, but little is said of electrical theory incomprehensible to the average operating surgeon. It should be emphasized that this is not just another volume on the extirpation of malignancies, but rather a treatise on the technique of skin, oral, otolaryngological, thyroid, breast, abdominal, urological operations, etc.

This book is the authoritative work on operative electrosurgery at the present time.

JOHN D. ELLIS.

THE seventh volume of this stupendous third edition[4] of Veit's *Handbuch der Gynaekologie* is in keeping with those volumes previously reviewed. It is encyclopedic in character devoting, as it does, almost six hundred pages to diseases of the ovary and parovarium, and over four hundred pages to tumors of the uterine tubes.

The first section is written by Kermauner of Vienna and is probably one of the last of his writings as his death occurred some months ago. This section is carefully and completely written and illustrated. It takes up every type of ovarian disease and ovarian and parovarian tumor that has ever been described in the world literature. It is well illustrated and contains many color plates. This section of the book makes a perfect reference work on the subjects under consideration.

The second half of the book is devoted to tumors of the uterine tubes. This subject is one of interest

[1] DISEASES OF THE THYROID GLAND: WITH SPECIAL REFERENCE TO THYROTOXICOSIS. By Cecil A. Joll, M.S., B.Sc. (Lond.), F.R.C.S. (Eng.). St. Louis: The C. V. Mosby Company, 1932.

[2] CHILDREN'S TONSILS IN OR OUT: A CRITICAL STUDY OF THE END-RESULTS OF TONSILLECTOMY. By Albert D. Kaiser, M.D. Philadelphia, London, Montreal: J. B. Lippincott Company, 1932.

[3] ELECTROSURGERY. By Howard A. Kelly, M.D., LL.D., F.A.C.S., and Grant E. Ward, M.D., F.A.C.S. Philadelphia and London: W. B. Saunders Company, 1932.

[4] VEIT'S HANDBUCH DER GYNAEKOLOGIE. Edited by Dr. W. Stoeckel. Vol. vii.—Die Erkrankungen der Eierstoecke und Nebeneierstoecke und die Geschwuelste der Eileiter. Edited by F. Kermauner and L. Nuernberger. Munich: J. F. Bergmann, 1932.

and must of course be included in any work dealing with gynecology. To devote over four hundred pages to this subject seems most unnecessary. This section is a complete résumé of all tumors of the uterine tubes reported to date which is desirable in an encyclopedic work of this type. Devoting two hundred pages to a tabulated analysis of the reports on primary carcinoma of the tubes seems to overshoot the purpose of this work. Nuernberger would have made his section much more readable and usable if he had reduced this section to approximately one-third or one-quarter of its present size.

RALPH A. REIS.

IT is very welcome to have the unsolved problem of idiopathic steatorrhœa presented in monographic form.[1] The solution of the etiology of sprue is especially fascinating since one feels that important physiological facts, hitherto unsuspected, must be brought to light with it. There is, of course, no such interest in this disease for those who consider sprue as definitely caused by monilia or for those who believe non-tropical sprue to be a rarity unrelated to tropical sprue. But the writer demonstrates from the cases studied here that this disease when recognized is the result of marked physiological disturbances, i.e., a functional disorder of unknown gastro-intestinal mechanisms; that minor grades of it are probably of frequent occurrence, and that tropical and non-tropical sprue are the same disease. This monograph is careful and scientific; photomicrographs of pathological material are included. The literature has been carefully studied and is reviewed constantly in connection with every aspect of the disease. PAUL STARR.

THE principles of elementary anatomy, physiology, massage, and remedial gymnastics are embodied in concise form in the excellent textbook[2] for students of massage by Despard. The anatomic illustrations are excellent, and the directions for massage and therapeutic exercise are clear and concise. It can be recommended as a textbook for physical therapy technicians. J. S. COULTER.

[1] NON-TROPICAL SPRUE; A STUDY IN IDIOPATHIC STEATORRHŒA. By Th. E. Hess Thaysen, M.D. Copenhagen: Levin & Munksgaard, London: Oxford University Press, 1932.

[2] TEXT BOOK OF MASSAGE AND REMEDIAL GYMNASTICS. 3d ed. By L. L. Despard. London and New York: Humphrey Mulford, Oxford University Press, 1932.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

NEUROLOGICAL EFFECTS OF SYPHILIS; DIAGNOSIS AND TREATMENT. By Bryan Buckley Sharp, M.D., M.R.C.P. (Lond.). London: Oxford University Press, 1933.

THE INTERNATIONAL MEDICAL ANNUAL. Editors: Carey F. Coombs, M.D., F.R.C.P. (the late), A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S. 1933, Fifty-First Year. Baltimore: William Wood and Co., 1933.

LYMPHATICS, LYMPH AND TISSUE FLUID. By Cecil K. Drinker, B.S., M.D., and Madeleine E. Field, A.B., Ph.D. Baltimore: The Williams & Wilkins Company, 1933.

SURGICAL PATHOLOGY. By William Boyd, M.D., M.R.C.P. (Ed.), F.R.C.P. (Lond.), etc. 3d ed. Philadelphia and London: W. B. Saunders Company, 1933.

DIE BLUTTRANSFUSION IN THEORIE UND PRAXIS. By Dr. Med. Hans Wildegans. Berlin: Julius Springer, 1933.

HOLT'S DISEASES OF INFANCY AND CHILDHOOD, etc. Revised by L. Emmett Holt, Jr., M.D., and Rustin McIntosh, M.D. 10th ed. New York and London: D. Appleton and Company, 1933.

INTERNAL DERANGEMENTS OF THE KNEE-JOINT, etc. By A. G. Timbrell Fisher, M.C., M.B., Ch.B., F.R.C.S. (Eng.). 2d ed. New York: The Macmillan Company, 1933.

ARTERIOSCLEROSIS; A SURVEY OF THE PROBLEM. (By various contributors.) A Publication of The Josiah Macy, Jr. Foundation. Edited by Edmund V. Cowdry. New York: The Macmillan Company, 1933.

THE RHYTHM OF STERILITY AND FERTILITY IN WOMEN. By Leo J. Latz, A.B., M.D., LL.D. 3d rev. ed. Chicago: Latz Foundation, 1933.

MEDICAL STATE BOARD EXAMINATIONS; TOPICAL SUMMARIES AND ANSWERS; REVIEW OF ACTUAL QUESTIONS GIVEN, etc. By Harold Rypins, A.B., M.D. Philadelphia. J. B. Lippincott Co., 1933.

THE CYCLOPEDIA OF MEDICINE. George Morris Piersol, B.S., M.D., Editor-in-Chief. Charles E. de M. Sajous, M.D., LL.D., Sc.D., Founder and First Editor. Vols. i–vii. Philadelphia: F. A. Davis Company, 1933.

ATLANTE DI CHIRURGIA DELLO STOMACO. By G. Egidi. Rome: Luigi Pozzi, 1933.

LE NYSTAGMUS VESTIBULAIRE ET LES RÉACTIONS DE MOUVEMENTS. By R. Claoué. Paris: N. Maloine, 1933.

NATURAL CHILDBIRTH. By Grantly Dick Read, M.A., M.D. London: William Heinemann, Ltd., 1933.

SURGICAL ANATOMY. By C. Latimer Callander, A.B., M.D., F.A.C.S. Philadelphia and London: W. B. Saunders Company, 1933.

MASSAGE AND REMEDIAL EXERCISES; IN MEDICAL AND SURGICAL CONDITIONS. By Noël M. Tidy. Baltimore: William Wood and Company, 1933.

DISEASES OF INFANCY AND CHILDHOOD. By Leonard G. Parsons, M.D., F.R.C.P., and Seymour Barling, C.M.G., F.R.C.S. New York, London: Oxford University Press, 1933.

THE WELLCOME RESEARCH INSTITUTION AND THE AFFILIATED RESEARCH LABORATORIES AND MUSEUMS. London: The Wellcome Foundation, Ltd., 1933.

A TEXT-BOOK OF NEUROPATHOLOGY. By Arthur Weil, M.D. Philadelphia: Lea & Febiger, 1933.

SURGERY OF THE STOMACH AND DUODENUM. By J. Shelton Horsley, M.D., F.A.C.S., LL.D. St. Louis: The C. V. Mosby Company, 1933.

MODERN ASPECTS OF GASTRO-ENTEROLOGY. By M. A. Arafa, M.R.C.P. (Lond.). Baltimore: William Wood and Company, 1933.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

J. BENTLEY SQUIER, New York, *President* WILLIAM D. HAGGARD, Nashville, *President-Elect*
FRANKLIN H. MARTIN, Chicago, *Director-General*
PHILIP H. KREUSCHER, *Chairman;* OSCAR E. NADEAU, *Secretary, Committee on Arrangements*

PRELIMINARY PROGRAM FOR THE CLINICAL CONGRESS IN CHICAGO

THE surgeons of Chicago are keenly interested to present a program of clinics and demonstrations that will provide a complete showing of the clinical activities in all departments of surgery in this great medical center during the twenty-third annual Clinical Congress of the American College of Surgeons, October 9–13. A preliminary schedule of operative clinics and demonstrations, as prepared by the Committee on Arrangements, is presented in the following pages. It will be noted that clinics are scheduled to begin at 2 o'clock on the afternoon of Monday, October 9, continuing through the four following days with sessions both morning and afternoon.

The Committee on Arrangements, under whose supervision the clinical program is being prepared, appointed by the Board of Regents of the College, is comprised of the members of an Executive Committee — Philip H. Kreuscher, chairman; Oscar E. Nadeau, secretary; Joseph Beck, William R. Cubbins, Frederick H. Falls, Harry S. Gradle, Carl A. Hedblom, Charles E. Kahlke, Herman L. Kretschmer, Karl A. Meyer, Dallas B. Phemister, Edwin W. Ryerson, and Henry Schmitz—together with representatives of each of the hospitals and medical schools appearing in the clinical program. In making its plans, the Committee has been assured of the hearty co-operation of the clinicians of the medical schools and more than fifty hospitals that will participate in the clinical program.

Special features of the clinical program include: (1) Cancer clinics demonstrating the treatment of cancer cases by surgery, radium and X-ray; (2) fracture clinics in several hospitals where modern methods of the treatment of fractures will be demonstrated; (3) traumatic surgery clinics demonstrating the newer methods of rehabilitation by surgery and physiotherapy of patients injured in industrial, automobile and other accidents.

Other important features of the general program for the Congress include: (1) Conference on fractures on Tuesday afternoon, arranged by the College Committee on the Treatment of Fractures, (2) a symposium under the auspices of the Board on Industrial Medicine and Traumatic Surgery on Friday afternoon, (3) a symposium on the teaching of surgery and the surgical specialties on Thursday afternoon following the annual meeting; (4) a symposium on urological surgery on Friday morning, the program for which appears on another page.

Two sub-committees have been appointed to supervise the program for the sections on surgery of the eye, ear, nose and throat as follows: Ophthalmology—Harry S. Gradle, chairman; Thomas D. Allen, E. K. Findlay, Sanford Gifford. Otolaryngology—Joseph Beck, chairman; Austin A. Hayden, Edward P. Norcross, S. J. Pearlman. The recommendations of these committees insure a worth-while program of clinics and scientific sessions for all those interested in these specialties.

The College celebrates its twentieth anniversary at this session—the first convocation having been held in Chicago in 1913. The first Clinical Congress was held in this city in 1910, and it will be recalled that that session was attended by a large number of enthusiastic surgeons from all parts of the United States and Canada.

EVENING MEETINGS

The Central Executive Committee of the Congress is preparing programs for a series of five evening meetings which are to be held in the ballroom of the Stevens Hotel. A preliminary outline of these programs will be found on another page.

An interesting feature of the presidential meeting on Monday evening, in addition to the inauguration of new officers, will be the introduction of distinguished visiting surgeons from foreign countries, a large number of whom have been especially invited to attend the Congress this year. Among those who have indicated their intention of being present are Prof. Dr. Eugen Kisch, Graefenberg, Bohemia; Mr. Adams A. McConnell, Dublin, Ireland; Prof. Nissen, Berlin, Germany; Prof. Dr. Wolfgang Rosenthal, Leipzig, Germany; Prof. H. Beckwith Whitehouse, Birmingham, England.

Programs are being prepared for sessions on Tuesday and Thursday evenings at which papers and discussions will deal with subjects of special interest to ophthalmologists and otolaryngologists.

SYMPOSIUM: CANCER IS CURABLE

In this symposium, to be held in the ballroom of the Stevens Hotel on Wednesday afternoon at 2:30, a large number of eminent surgeons of wide experience in varied fields of surgical practice, from all parts of the United States and Canada, will present their reports as to cases of cancer cured for a period of five years or longer. At the Congress in St. Louis last year a similar symposium established a new viewpoint for the profession and the laity, creating widespread favorable comment.

Cancer as an Arrestable Disease: ROBERT B. GREENOUGH, M.D., Boston, Chairman, Committee on the Treatment of Malignant Diseases; FRANKLIN H. MARTIN, M.D., Director-General, American College of Surgeons; CHARLES A. DUKES, M.D., Oakland, Calif.

General Cases of Five-Year Cures: IRVIN ABELL, M.D., Louisville, Ky.; Frank K. BOLAND, M.D., Atlanta, Ga.; JOHN JOSEPH GALLIGAN, M.D., Salt Lake City, Utah; CHARLES C. LUND, M.D., Boston; JAMES MONROE MASON, M.D., Birmingham, Ala.; JOHN T. MOORE, M.D., Houston; DAMON B. PFEIFFER, M.D., Philadelphia; EUGENE H. POOL, M.D., and J. A. VIETOR, M.D., New York.

Cancer of the Breast: MALVERN B. CLOPTON, M.D., St. Louis; E. STARR JUDD, M.D., and S. W. HARRINGTON, M.D., Rochester, Minn.; RICHARD R. SMITH, M.D., Grand Rapids, Mich.

Cancer of the Pelvic Organs and Breast: BROOKE M. ANSPACH, M.D., Philadelphia; HARRY S. CROSSEN, M.D., St. Louis; WILLIAM P. HEALY, M.D., New York.

Cancer of the Pelvic Organs: JAMES C. MASSON, M.D., Rochester, Minn.

Cancer of the Rectum: ROBERT C. COFFEY, M.D., Portland, Ore.

Cancer of the Thyroid Gland and Large Intestine: JOHN DEJ. PEMBERTON, M.D., and C. F. DIXON, M.D., Rochester, Minn.

Cancer of the Thyroid: MARTIN B. TINKER, M.D., Ithaca, N.Y.

Cancer of the Mouth, Tongue and Lips: WILLIAM H. G. LOGAN, M.D., Chicago.

Malignant Bone Tumors: WILLIAM B. COLEY, M.D., New York.

Cancer of the Throat, Œsophagus and Bronchi: CHEVALIER JACKSON, M.D., Philadelphia.

ANNUAL HOSPITAL CONFERENCE

An interesting program of papers, round table conferences and practical demonstrations dealing with the many problems related to hospital efficiency is being prepared for the sixteenth annual hospital conference which opens at 10 o'clock on Monday morning in the ballroom of the Stevens Hotel, continuing on Tuesday, Wednesday and Thursday. It is planned to give the program a broad interest with a careful selection of subjects to be discussed by eminent authorities in the surgical and hospital field. The papers will deal with many of the vital problems affecting administrative, professional and nursing phases of hospital work. Particular emphasis will be directed toward professional standards and the highly important problem of medical economics.

Morning sessions on Tuesday, Wednesday and Thursday will be devoted to papers, discussions and round table conferences at the Stevens Hotel. An important and most interesting feature this year will be a series of demonstrations in several of the hospitals on Tuesday, Wednesday and Thursday afternoons dealing with departmental organization, management and functions. These "clinics" in hospital administration will afford opportunities for the visitors to see how others do the things they are constantly doing in their own institutions, and in comparison appraise their own methods.

The greatly increased interest on the part of the surgeon in both the administrative and scientific phases of hospital work has been evidenced in recent years, and for this reason the program to be presented will be unique in providing a discussion of several subjects of importance to the three major groups working in the hospital—medical, nursing and business. An opportunity will be afforded to chiefs of staffs, heads of departments and members of medical staffs to participate in a program that deals particularly with the care of the patient, expecting to benefit by contact with and interchange of ideas with trustees, superintendents and others concerned with hospital administration.

HEADQUARTERS—HOTELS

General headquarters for the Clinical Congress will be established at the Stevens Hotel, located on Michigan Avenue between Seventh and Eighth Streets. This hotel affords unusual facilities for

all activities of the Congress, as will be remembered by those who attended the Congress in Chicago in 1929. The grand ballroom on the second floor with other large rooms on the third floor and the exhibition hall have been reserved for the exclusive use of the Congress. All of the evening sessions, the hospital conference on Monday, the annual meeting, the cancer and fracture symposia will be held in the grand ballroom. The registration and information bureau, together with the bulletin boards on which will be displayed the daily clinical program will be established in the exhibition hall in the basement, together with the Technical Exhibition.

Chicago has many fine, large hotels, several within walking distance of the headquarters hotel. A list of the hotels recommended by the Committee on Arrangements is presented herewith. While Chicago's hotel facilities are very great and there should be no difficulty in securing first class hotel accommodations, it is advisable for those who expect to attend the Clinical Congress to reserve their hotel accommodations as far in advance as possible, as the Century of Progress Exposition will undoubtedly bring to Chicago a very large number of visitors.

The Technical Exhibition of the Clinical Congress will be located in the Exhibition Hall together with the registration and information bureau. In the same room will be found the bulletin boards on which the daily clinical programs will be posted each afternoon. The leading manufacturers of surgical instruments, X-ray apparatus, operating room lights, hospital apparatus and supplies of all kinds, ligatures, dressings, pharmaceuticals, and publishers of medical books will be represented in this exhibition.

We are assured that the railways of the United States and Canada will grant especially low rates on account of the Clinical Congress in connection with the Century of Progress Exposition in Chicago. Applications for reduced fares are pending before the railway traffic associations.

ADVANCE REGISTRATION

The hospitals of Chicago afford accommodations for a large number of visiting surgeons, but to insure against overcrowding, the attendance will be limited to a number that can be comfortably accommodated at the clinics—the limit of attendance being based upon the results of a survey of the amphitheaters, operating rooms, and laboratories of the hospitals and medical schools to determine their capacity for visitors. It is expected, therefore, that those surgeons who wish to attend the Clinical Congress in Chicago will register in advance.

Attendance at all clinics and demonstrations will be controlled by means of special clinic tickets, which plan provides an efficient means for the distribution of the visiting surgeons among the several clinics and insures against overcrowding, as the number of tickets issued for each clinic will be limited to the capacity of the room in which that clinic will be given.

A registration fee of $5.00 is required of each surgeon attending the annual Clinical Congress, such fees providing the funds with which to meet the expenses of the meeting. To each surgeon registering in advance a formal receipt for the registration fee is issued, which receipt is to be exchanged for a general admission card upon his registration at headquarters. This card, which is non-transferable, must be presented in order to secure clinic tickets and admission to the evening meetings.

CHICAGO HOTELS AND THEIR RATES

	Minimum Rates With Bath	
	Single	Double
Ambassador, North State Street at Goethe	$3.50	$6.00
Auditorium, Michigan Blvd. and Congress	3.50	6.00
Belden Stratford, 2300 Lincoln Park West	4.00	6.00
Belmont, Sheridan Road at Belmont	4.00	5.00
Bismarck, Randolph at LaSalle St.	3.50	5.00
Blackstone, Michigan Blvd. and 7th St.	3.00	5.00
Brevoort, 120 West Madison St.	2.50	3.50
Congress, Michigan Blvd. and Congress	4.00	6.00
Drake, Lake Shore Drive and Michigan	3.00	5.00
Edgewater Beach, 5300 Sheridan Road	4.00	6.00
Great Northern, Jackson and Dearborn	2.50	4.00
Knickerbocker, 163 East Walton	3.00	5.00
LaSalle, LaSalle at Madison St.	2.50	4.00
Morrison, 79 West Madison St.	3.00	4.50
Palmer House, State and Monroe Sts.	3.50	6.00
Pearson, 190 East Pearson St.	3.00	5.00
Stevens, Michigan Blvd. bet. 7th and 8th	3.50	5.00

PRELIMINARY PROGRAM FOR EVENING MEETINGS

IN THE BALLROOM OF THE STEVENS HOTEL AT 8:15

Presidential Meeting, Monday, October 9

Address of Welcome. PHILIP H. KREUSCHER, M.D., Chairman of Committee on Arrangements
Introduction of Foreign Guests. FRANKLIN H. MARTIN, M.D., Director-General
Address of Retiring President. J. BENTLEY SQUIER, M.D., New York
Inauguration of Officers
Inaugural Address. WILLIAM D. HAGGARD, M.D., Nashville, Tenn.
John B. Murphy Oration in Surgery. LOYAL DAVIS, M.D., Chicago

Tuesday, Wednesday and Thursday, October 10, 11 and 12

Symposium on Vascular Diseases:
- Thrombo-Angiitis Obliterans (Buerger's Disease). GEORGE E. BROWN, M.D., Rochester, Minn.
- Ligation of Large Arteries. MONT ROGERS REID, M.D., Cincinnati

Symposium on Diseases of the Thyroid:
- Hyperthyroidism and Associated Diseases. GEORGE W. CRILE, M.D., Cleveland
- The Treatment of Exophthalmos. HOWARD C. NAFFZIGER, M.D., San Francisco
- Tumors of the Parathyroid Glands. EDWARD D. CHURCHILL, M.D., Boston

The Common Syndrome of Rupture, Dislocation and Elongation of the Biceps Brachii; an Analysis of Over Forty Cases. EDGAR L. GILCREEST, M.D., San Francisco
Sympathectomy in Children. DAVID EDWIN ROBERTSON, M.D., Toronto

Convocation—Friday, October 13

Invocation
Conferring of Fellowships
Conferring of Honorary Fellowships
Presidential Address. WILLIAM D. HAGGARD, M.D., Nashville, Tenn.
Fellowship Address. ROBERT MAYNARD HUTCHINS, A.M., LL.D., President, University of Chicago

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SYMPOSIUM ON UROLOGICAL SURGERY

BALLROOM, STEVENS HOTEL, FRIDAY, 11 A.M.

JOHN R. CAULK, M.D., St. Louis: Transurethral Surgery.
FRANK HINMAN, M.D., San Francisco: The Pathogenesis of Hydronephrosis.
JOSEPH F. MCCARTHY, M.D., New York: The Prostate Gland—Its Place in General Medicine; Newer Conception of Diagnosis and Therapy.

PRELIMINARY CLINICAL PROGRAM

GENERAL SURGERY, GYNECOLOGY, OBSTETRICS, ORTHOPEDICS, UROLOGY, PROCTOLOGY, SURGICAL PATHOLOGY, ETC.

PASSAVANT MEMORIAL HOSPITAL—NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

Tuesday

LEANDER W. RIBA—9. The use of the electro-urethrotome in urethral strictures.
ARTHUR H. CURTIS and GEORGE H. GARDNER—9. Gynecological operations.
JOHN A. WOLFER—9. Cholecystitis, carcinoma of colon.
JACOB R. BUCHBINDER—9. Thyroid surgery.
JOHN S. COULTER—10. Physical therapy.
RUDOLPH W. HOLMES and staff—2. Symposium on cardiac diseases in their obstetric associations. CHAUNCEY C. MAHER: Etiology and pathology. JAMES E. FITZGERALD: Medical aspects and treatment. JAMES H. BLOOMFIELD: Obstetrical aspects and treatment.
PAUL B. MAGNUSON—2. Ununited fracture of the neck of the femur, bone graft in the spine.
JOHN A. WOLFER—2. Dry clinic: Alimentation of the critically ill patient by jejunal feedings.
LOYAL DAVIS, LEWIS J. POLLOCK, HALE HAVEN and DAVID A. CLEVELAND—2. Symposium on neurologic surgery.

Wednesday

HARRY M. RICHTER—9. Thyroid surgery.
LOYAL DAVIS—9. Neurologic surgery.
SUMNER L. KOCH and MICHAEL L. MASON—9. Nerve and tendon surgery of the hand.
JAMES T. CASE—10. Roentgenology.
PHILIP H. KREUSCHER—2. Hip joint surgery.
ALLEN B. KANAVEL, SUMNER L. KOCH and M. L. MASON—2. Review of twenty years of surgery of the hand.
RUDOLPH W. HOLMES and staff—2. Symposium on toxæmias of late pregnancy, renal and hepatic. JAMES P. SIMONDS: Etiology and pathology. CHESTER C. DOHERTY: Symptoms and laboratory investigation. DAVID S. HILLIS: Medical (expectant) treatment. RUDOLPH W. HOLMES: Obstetrical treatment.
LEANDER W. RIBA—2. Dry clinic: Prostatic resection.
EMIL D. W. HAUSER—2. Orthopedic surgery.

Thursday

ARTHUR H. CURTIS and GEORGE H. GARDNER—9. Gynecological operations.
JOHN A. WOLFER—9. Cholecystitis; carcinoma of the breast.
JACOB R. BUCHBINDER—9. Abdominal surgery.
JOHN S. COULTER—10. Physical therapy.
PHILIP H. KREUSCHER—2. Shoulder and knee-joint derangement.
RUDOLPH W. HOLMES and staff—2. Symposium on obstetrical hæmorrhages. RUDOLPH W. HOLMES: Ablatio placentæ. DAVID S. HILLIS: Placenta prævia. MAGNUS P. URNES: Postpartum hæmorrhages. THEODORE W. BLUECHEL: Treatment of sequential anæmias.
CHARLES A. ELLIOTT, WALTER H. NADLER, PAUL STARR, M. HERBERT BARKER, HOWARD B. CARROLL and HOWARD L. ALT—2. Symposium on hepatic disease.

Friday

HARRY M. RICHTER—9. Gastric surgery.
LOYAL DAVIS—9. Neurologic surgery.
SUMNER L. KOCH and MICHAEL L. MASON—9. Irradiation ulcers of the hand, Dupuytren's contracture.
JAMES T. CASE—10. Roentgenology.
PAUL B. MAGNUSON—2. Demonstration of principles for overcoming deformity in ununited fractures before operation; bone grafts for ununited fractures.
RUDOLPH W. HOLMES and staff—2. Symposium on hyperemesis gravidarum. CHESTER C. DOHERTY: Etiology and pathology. MAGNUS P. URNES: Symptoms and clinical course. JAMES H. BLOOMFIELD: Treatment.
HARRY M. RICHTER, ANDREW C. IVY, SAMUEL J. FOGELSON and A. J. ATKINSON—2. Symposium on gastric ulcer.

MOUNT SINAI HOSPITAL

Tuesday

V. L. SCHRAGER and associates—9. Hernia, breast and biliary surgery.
ISRAEL DAVIDSOHN—11. Pathological demonstration.
M. I. KAPLAN—11. X-ray diagnosis and therapy.
GUSTAV KOLISCHER and HARRY ROLNICK—2. Genito-urinary surgery.

Wednesday

HARRY M. RICHTER and associates—9. Gastric and thyroid surgery.
ISRAEL DAVIDSOHN—11. Pathological demonstration.
M. I. KAPLAN—11. X-ray diagnosis and therapy.
ALFRED A. STRAUSS—2. Gastro-intestinal surgery.
RALPH B. BETTMAN and associates—2. Intrathoracic surgery, operations.

Thursday

AARON KANTER, A. F. LASH and associates—9. Gynecological operations.
ISRAEL DAVIDSOHN—11. Pathological demonstration.
M. I. KAPLAN—11. X-ray diagnosis and therapy.
CHARLES JACOBS and associates—2. Orthopedic operations.

Friday

HARRY ROLNICK—9. Genito-urinary surgery.
ISRAEL DAVIDSOHN—11. Pathological demonstration.
M. I. KAPLAN—11. X-ray diagnosis and therapy.

Dry Clinics—Daily, 9 and 2

ISRAEL DAVIDSOHN. Value of biopsy in surgery.
HENRY BUXBAUM. Toxæmias of pregnancy.
GUSTAV KOLISCHER. Electrosurgery in cancer therapy.
AARON KANTER. Chorio-epithelioma following a vesicular mole; functional uterine hæmorrhage.
HARRY ROLNICK. Bladder tumors.
A. F. LASH. Treatment of birth injury; early diagnosis of uterine cancer.
DAVID A. WILLIS. Relation of adrenals to thyrotoxicosis; morbidity in operation for acute appendicitis in relation to the question of drainage; demonstration of a universal traction splint as used in a small hospital.
EMIL L. AISON. Fractures of the maxilla and mandible.
M. REESE GUTTMAN. Recent advances in the treatment of malignant diseases about the head and neck; endoscopic clinic.
Staff. Symposium: Cancer of the lung. I. M. TRACE, medical aspect; JACOB LIFSCHUTZ, bronchoscopic aspect; ISRAEL DAVIDSOHN, pathological aspect; M. I. KAPLAN, X-ray aspect.
MAURICE LEWISON. Medical appraisal of surgical risks.

PRESBYTERIAN HOSPITAL

Tuesday

A. D. BEVAN—9. Surgery of the breast.
V. C. DAVID—9. Carcinoma of sigmoid.
H. L. KRETSCHMER—9. Kidney surgery.
R. H. HERBST—9. Transurethral electro resection of prostate gland.
KELLOGG SPEED—9. Tumors of chest wall, demonstration of cases, lantern slides.
A. H. MONTGOMERY—11. Abdominal surgery in children.
A. VERBRUGGHEN—2. Neurosurgical operation.

Wednesday

A. D. BEVAN—9. Hernia and undescended testicle.
F. B. MOOREHEAD—9. Plastic surgery of mouth and face
C. B. DAVIS—9. Tumors of the large intestine.
H. L. KRETSCHMER—9. Surgery of the bladder.
N. S. HEANEY—9. Vaginal surgery.
DR. GATEWOOD—10. Carcinoma of the stomach, follow-up clinic.
E. M. MILLER—10. Thyroid surgery.
H. A. OBERHELMAN—10. Surgery in diabetic patients.
E. R. MCCARTHY—11. Strangulated hernia in infants.
W. J. POTTS—12. Fracture problems.

Thursday

A. D. BEVAN—9. Surgery of gall bladder and bile tract.
H. L. KRETSCHMER—9. Transurethral resection of the prostate.
F. B. MOOREHEAD—9. Cleft palate surgery; operative treatment of ankylosis of jaw.
DR. GATEWOOD—9. Gastric resection for ulcer.
R. H. HERBST—9. Diverticula of urinary bladder.
R. H. HERBST and C. W. APFELBACH—9. Unusual urinary anomalies.
Staff—9. Dry clinic: E. D. ALLEN, endometriosis; C. P. BAUER, dystocia; AARON KANTER, recognition of early carcinoma of uterus.
G. L. MCWHORTER—10. Fracture of the greater tuberosity of the humerus.
A. VERBRUGGHEN—10. Spinal cord injuries.

Friday

Staff—9. Dry clinic: A. D. BEVAN. Present status of anæsthesia. H. L. KRETSCHMER. Genito-urinary surgery. R. C. BROWN. Treatment of massive hæmorrhage in gastric ulcer. V. C. DAVID. Significance of polyps of large bowel. E. M. MILLER. Method of intravenous injection over long period of time. R. H. HERBST. Fibrosis of bladder neck. F. H. STRAUS. Obstructive jaundice. G. L. MCWHORTER. Reconstruction of common bile duct, cases. M. L. LORING. Granuloma inguinale, cases. S. E. LAWTON. Cholecystenterostomy, indications.
E. J. BERKHEISER—2. Orthopedic clinic.

WASHINGTON BOULEVARD HOSPITAL

Tuesday

PAUL C. FOX—9. Gynecological clinic.

Wednesday

A. R. METZ—9. General surgical clinic, presentation of unusual fractures.

Thursday

V. J. O'CONOR—9. Hydronephrosis, etiology and treatment, case reports, X-rays and operative results; suprapubic prostatectomy and transurethral resection of prostate comparatively, indications and results.

ST. LUKE'S HOSPITAL

Monday

H. E. MOCK, A. REID MORROW and CHARLES SHANNON—2. General surgical operations.
E. OLDBERG—2. Neurological surgery.

Tuesday

H. O. JONES, WILLIAM P. CARLISLE, M. J. KILEY, E. A. EDWARDS and JOHN BREWER—9. Gynecological operations; early human embryo, demonstration.
CARL HEDBLOM and WILLARD VAN HAZEL—9. Thoracic surgery.
H. E. MOCK—2. Reconstructive surgery.
L. L. MCARTHUR and S. W. MCARTHUR—2. General surgery.

Wednesday

L. E. SCHMIDT—9. Urological clinic.
E. W. RYERSON and F. A. CHANDLER—9. Orthopedic operations.
S. C. PLUMMER—9. General surgery.
H. E. JONES and T L. HANSEN—9. General surgery.
E. W. RYERSON, R. O. RITTER and H. O. SOFIELD—2. Orthopedic operations.
FRANK E. DAVID, C. J. DEBERE and G. V. PONTIUS—2. Rectal surgery.

Thursday

G. DETAKATS—9. Surgery in juvenile diabetes; ambulatory vein ligation of varicose veins.
H. E. MOCK—9. General surgery.
HARRY CULVER—9. Urological clinic.
H. E. MOCK, A. REID MORROW and CHARLES SHANNON—2. Skull fractures.
W. R. CUBBINS—2. General surgery.
H. B. THOMAS and F. W. HARK—2. Orthopedic clinic.

Friday

W. F. LYON—9. Dislocations of the shoulder with fracture of the greater trochanter.
H. POTTS and F. W. MERRIFIELD—9. Oral surgery, operative.
E. W. RYERSON, F. A. CHANDLER and R. O. RITTER—2. Orthopedic clinic.

SOUTH SHORE HOSPITAL

Tuesday

AXEL WERELIUS—9. Gastric surgery.
GEORGE G. O'BRIEN—11. General surgery.
CLARENCE S. DUNER and AXEL WERELIUS—2. Symposium on gastric and duodenal ulcer.

Wednesday

HUGH MACKECHNIE—9. Surgery of the colon.
FRANK G. MURPHY—11. Orthopedic clinic.
H. WILLIAM ELGHAMMER, GUY S. VAN ALSTYNE and PAUL R. CANNON—2. Symposium on intussusception.

Thursday

LOUIS D. SMITH—9. Genito-urinary surgery.
CLARA JACOBSON—2. Lung collapse procedures.
C. C. MAHER—3. Cardiac risk in surgery.

Friday

E. A. LUTTON—9. Gynecological clinic.
ANDREW DAHLBERG and WILLIAM HANRAHAN—11. Operative obstetrics.
H. R. COLVER—2. Industrial surgery.
WALTER FISCHER—3. Foot problems.

COOK COUNTY HOSPITAL

Monday

SUMNER L. KOCH—2. General surgery.
F. H. FALLS—2. Gynecology.
E. J. BERKHEISER—2. Orthopedics.
WILLIAM R. CUBBINS—2. General surgery.
M. DAVISON—2. General surgery.

Tuesday

SUMNER L. KOCH—9. Diagnostic clinic.
AARON KANTER—9. General surgery.
GEORGE DAVIS—9. General surgery.
A. H. MONTGOMERY—9. General surgery.
A. H. CONLEY—9. Orthopedics.
CAREY CULBERTSON—9. Gynecology.
J. O'DONOGHUE—9. General surgery.
HARRY CULVER—9. Urology.
H. JACKSON—9. General surgery.
MARCUS HOBART—9. Orthopedics.
VERNON C. DAVID—9. Diagnostic clinic.
L. C. GATEWOOD—9. General surgery.
J. P. GREENHILL—2. Gynecology.
RALPH B. BETTMAN—2. Surgery in tuberculosis.
E. WARSZEWSKI—2. General surgery.

Wednesday

CHANNING BARRETT—9. Gynecology.
HARRY CULVER—9. General surgery.
V. L. SCHRAGER—9. General surgery.
GEORGE APFELBACH—9. General surgery.
J. G. FROST—9. General surgery.
R. C. SULLIVAN—9. General surgery.
L. L. VESEEN—9. Urology.
FRANK JIRKA—9. General surgery.
R. VAUGHAN—9. General surgery.
PHILIP H. KREUSCHER—9. Orthopedics.
CHARLES M. MCKENNA—9. Urology.
H. ROLNICK—2. Urology.
HARRY CULVER—2. Urology.
GEORGE DAVIS—2. General surgery.
J. R. BUCHBINDER—2. General surgery.
DAVID HILLIS—2. Obstetrical operations.
SUMNER L. KOCH—2. General surgery.

Thursday

PHILIP H. KREUSCHER—9. Orthopedics.
CHANNING BARRETT—9. General surgery.
GEORGE DAVIS—9. General surgery.
R. W. MCNEALY—9. General surgery.
MARCUS HOBART—9. Orthopedics.
D. HORNER—9. Gynecology.
KARL A. MEYER—9. General surgery.
E. W. FISCHMAN—9. Gynecology.
A. H. MONTGOMERY—9. General surgery.
MAX THOREK—9. General surgery.
A. H. CONLEY—9. Orthopedics.
D. H. LEVINTHAL—9. Orthopedics.
JOHN HARGER—2. General surgery.
F. H. FALLS—2. Gynecology.
E. J. BERKHEISER—2. Orthopedics.
RALPH BETTMAN—2. General thoracic surgery.
WILLIAM R. CUBBINS—2. General surgery.

Friday

GEORGE APFELBACH—9. General surgery.
AARON KANTER—9. General surgery.
R. C. SULLIVAN—9. General surgery.
CAREY CULBERTSON—9. Gynecology.
VERNON C. DAVID—9. General surgery.
MARCUS HOBART—9. Orthopedics.
F. G. DYAS—9. General surgery.
J. O'DONOGHUE—9. General surgery.
H. JACKSON—9. General surgery.
L. C. GATEWOOD—9. General surgery.
JOHN HARGER—2. General surgery.
J. R. BUCHBINDER—2. General surgery.
MARSHALL DAVISON—2. General surgery.
E. WARZEWSKI—2. General surgery.
SUMNER L. KOCH—2. General surgery.

MERCY HOSPITAL

Tuesday

E. M. BROWN—9. Malignancy of the colon.
J. E. KELLY—9. Chronic intestinal fistula; extensive ventral hernia.
GEORGE GRIFFIN—9. Pyloric obstruction.
J. D. CLARIDGE—9. Fractures and dislocations of the cervical spine.
C. J. LARKIN—9. Rupture of the spleen simulating acute appendicitis.

Wednesday

M. F. MCGUIRE—9. Biliary tract surgery.
C. F. SAWYER—9. Acute pancreatitis; perforating gastric and duodenal ulcers.
C. L. MARTIN—9. Anal fistulectomies in cases with pulmonary tuberculosis.
L. E. GARRISON—9. Carcinoma of the colon; carcinoma of the breast.
HERBERT E. LANDES—9. Surgical anatomy of vesical orifice and urethral obstructions; treatment of bladder tumors.

Thursday

L. D. MOORHEAD—9. Toxic goiters; differential diagnosis of cases of dysthyroidism and hyperthyroidism with indication for operation and management.
W. J. PICKETT—9. Technical considerations in posterior gastro-enterostomy.
F. E. PIERCE—9. Fracture cases.
F. M. DRENNAN and F. C. VALDEZ—9. Gastro-intestinal clinic.

Friday

HENRY SCHMITZ and HERBERT E. SCHMITZ—9. Gynecological clinic; surgery and radiation therapy.
JOSEPH LAIBE—9. Carcinoma of the genito-urinary tract.
A. M. VAUGHN—9. Cystic hygroma in an infant.

CHILDREN'S MEMORIAL HOSPITAL

Monday

FREMONT A. CHANDLER, CHARLES N. PEASE and FERDINAND SEIDLER—2. Orthopedic clinic.

Tuesday

FREMONT A. CHANDLER, FERDINAND SEIDLER and CHARLES N. PEASE—9. Orthopedic operations.
FREDERICK B. MOOREHEAD—2. Oral surgery, operations and demonstration of cases.

Wednesday

ALBERT H. MONTGOMERY and staff—9. General surgery, operations and demonstration of cases.

Thursday

HERMAN L. KRETSCHMER and staff—9. Urological surgery, operations and demonstration of cases.

Friday

ALBERT H. MONTGOMERY and staff—9. General surgery, operations and demonstration of cases.

MICHAEL REESE HOSPITAL

Tuesday

ALFRED A. STRAUSS, SIEGFRIED F. STRAUSS, JAMES PATEDJL and ROBERT A. CRAWFORD. Stomach resections for gastric and duodenal ulcer; common duct duodenal anastomosis and gastro-enterostomy for chronic obstructive jaundice.

GEORGE L. DAVENPORT and RALPH BETTMAN. Gall-bladder surgery; surgery of the common duct.

D. C. STRAUSS. Thyroid surgery.

E. FRIEND. General surgery; surgery of the gall bladder.

BERNARD PORTIS. Thyroid surgery; surgery of the rectum.

HARRY RICHTER. Thyroid surgery; gall-bladder surgery.

MAX CUTLER. Surgery of the breast.

GUSTAV KOLISCHER. Diathermy of bladder tumor; nephrectomy for tuberculosis.

IRVING KOLL. Electrical resection of prostate; nephrolithotomy.

DANIEL H. LEVINTHAL. Internal derangements of the knee joint; removal of semi-lunar cartilage; synovectomy for chronic arthritis; bone lengthening operation.

JULIUS E. LACKNER. Abdominal hysterectomy; interposition operation; rectovaginal fistula.

JOSEPH L. BAER and RALPH REIS. Complete perineal laceration, ovarian tumor and pelvic inflammation.

Wednesday

D. C. STRAUSS. Thyroid surgery; gall-bladder surgery.

RALPH BETTMAN. Surgery of the chest.

GEORGE L. DAVENPORT. General surgery.

ALFRED A. STRAUSS, SIEGFRIED F. STRAUSS and ROBERT A. CRAWFORD. Sectional colectomy for ulcerative colitis and pyloroplasty for congenital pyloric stenosis.

BERNARD PORTIS. General surgery and surgery of the colon.

MORRIS L. PARKER. General surgery.

JAMES PATEDJL. General surgery.

JOSEPH EISENSTAEDT. Undescended testis; suprapubic prostatectomy.

HARRY ROLNICK. Electric resection of prostate; pyelotomy for stones.

PHILIP LEWIN and SIDNEY SIDEMAN. Orthopedic clinic, shoulder, elbow, hand, hip, pelvis.

L. E. FRANKENTHAL, SR. and L. E. FRANKENTHAL, JR. Gynecological operations.

W. H. RUBOVITS. Obstetrical and gynecological clinic, demonstration of forceps, version and complete suture, episiotomy.

IRVING STEIN and M. L. LEVENTHAL. Obstetrical clinic, low cervical cæsarean under local anæsthesia.

Thursday

RALPH BETTMAN. Surgery of the gall bladder and common duct.

ALFRED A. STRAUSS, SIEGFRIED F. STRAUSS and ROBERT A. CRAWFORD. Surgical diathermy for carcinoma of the rectum; resections for carcinoma of the stomach.

D. C. STRAUSS. Surgery of the colon, small intestine and thyroid.

GEORGE L. DAVENPORT. Surgery of the common duct.

BERNARD PORTIS. General surgery.

SIEGFRIED F. STRAUSS. General surgery.

HARRY RICHTER. Surgery of the thyroid.

E. FRIEND. Surgery of the gall bladder and common duct.

ALFRED E. JONES. Nephrectomy for tuberculous kidney; suprapubic prostatectomy.

IRVING SHAPIRO. Diathermy of bladder tumor; nephrectomy for tumor of kidney.

DANIEL H. LEVINTHAL. Surgery of the spine, fusion operation for scoliosis and for tuberculosis.

CHARLES M. JACOBS. Orthopedic clinic.

JULIUS E. LACKNER. Gynecological operations.

JOSEPH L. BAER and RALPH REIS. Prolapse; vaginal hysterectomy; fibroids; occiput posterior.

Friday

ALFRED A. STRAUSS, SIEGFRIED F. STRAUSS, JAMES PATEDJL and ROBERT A. CRAWFORD. Subtotal gastrectomy for gastrojejunal ulcer; resection of colon for carcinoma.

D. C. STRAUSS. Surgery of the thyroid and general surgery.

GEORGE L. DAVENPORT and RALPH BETTMAN. Gall-bladder surgery and surgery of the common duct.

RALPH BETTMAN. Thoracic surgery.

BERNARD PORTIS. Surgery of the colon and rectum.

MORRIS L. PARKER. General surgery.

MAX CUTLER. Surgery of the breast; use of radiotherapy in carcinoma.

FREDERICK LIEBERTHAL. Suprapubic prostatectomy; ureterotomy.

J. S. GROVE. Undescended testes.

PHILIP LEWIN and SIDNEY SIDEMAN. Orthopedic clinic, back, hip, knee, foot, shoulder; demonstration of arthritis cases.

L. E. FRANKENTHAL, SR. and L. E. FRANKENTHAL, JR. Gynecological clinic.

W. H. RUBOVITS. Gynecological clinic.

IRVING STEIN and M. L. LEVENTHAL. Gynecological clinic.

RAVENSWOOD HOSPITAL

Tuesday

G. W. GREEN—9. Gall-bladder surgery, mortality and morbidity.

C. A. BUSWELL—9:30. Survey of cancer study organization in a private hospital.

D. B. POND—10. Orthopedic surgery.

E. W. MUELLER and J. J. MOORE—10:30. Carcinoma of testis.

M. FIELD—11. Diagnosis and management of sterility.

L. C. FRENCH and D. L. JENKINSON—11:30. Gastric syphilis.

Wednesday

G. DE TARNOWSKY and J. J. MOORE—9. Carcinoma of colon, modified Kraske operation.

J. IRELAND—9:30. Fractures of the elbow.

R. F. WEISSBRENNER—10. Emotions as etiological factors in hyperthyroidism.

C. H. LOCKWOOD—10:15. Headaches.

H. P. SAUNDERS—11. Blood transfusion.

L. E. DAY—11:15. Obstetrics.

J. F. OATES—11:30. Spinal anæsthesia.

Thursday

C. C. RENTFRO—9. Obstetrical anæsthesia.

W. F. GROSVENOR—9:15. Cæsarean section.

A. C. HAMMETT—9:30. Mental disturbances of diabetics.

A. V. BERGQUIST—9:45. Indigestion.

F. N. BUSSEY—10. Granulosa cell carcinoma of ovary.

R. E. DYER—10:30. Surgical technique.

P. J. SARMA—11. Paramedian abdominal incision.

F. R. VON NAHOWSKI—11:15. Mortality in appendicitis.

E. B. WILLIAMS—11:30. Perthes' disease; fracture of spine.

EVANGELICAL HOSPITAL

G. ERMAN JOHNSON. Clinical studies of extra-uterine pregnancy.

PERCY E. HOPKINS. Clinical studies of pancreatitis.

CHARLES PAPIK. Treatment of lower limb fractures by fixed traction.

PAUL GEORGE PAPSDORF. Demonstration of models and photographs showing newer methods of the handling of fractures of the maxilla and mandible.

ST. MARY OF NAZARETH HOSPITAL

Monday

A. S. SAMPOLINSKI—2. General surgical clinic.
E. H. WARSZEWSKI and P. F. CZWALINSKI—2. Inguinal hernia clinic.
THAD LARKOWSKI—2. Demonstration of blood transfusion.

Tuesday

GEORGE MUELLER—9. General surgical clinic.
S. R. PIETROWICZ—9. Spinal puncture and anæsthesia—indications, contra-indications, advantages, disadvantages, demonstrations.
C. C. BUCZYNSKI—2. Varicocele operations and demonstrations.
M. J. BADZMIEROWSKI and B. PIERZYNSKI—2. Goiter clinic, operations and demonstration of cases.

Wednesday

T. Z. XELOWSKI—9. Gynecology and abdominal surgery.
W. A. KUFLEWSKI—9. Emergency and general surgery.
THOMAS PLANT—9. General surgery.
A. A. THIEDA—9. General surgery.
FRANK TENZAR—9. General surgery.
JOHN TENZAR—9. General surgery.
CHESTER CHALLENGER—9. X-ray demonstration.
MICHAEL KUTZA—2. General surgery.
F. A. MACKOWIAK—2. General surgery.
M. E. UZNANSKI—2. Obstetrical clinic, low cæsarean section.
M. KRUPINSKI—2. Removal of pilanodal cyst.

Thursday

LEO CZAJA—9. Orthopedic clinic; maggot treatment of osteomyelitis.
E. MACDONALD—9. Abdominal surgery.
H. H. HILL—9. Demonstration of pathological specimens.
A. V. PARTIPILLO—2. Aseptic resection of the bowel, demonstration of cases, moving picture exhibition.
M. E. UZNANSKI—2. Surgical anatomy of the perineum, lantern slide demonstration.

Friday

JOSEPH WELFELD—9. Urological clinic.
GEORGE MUELLER—9. General surgery.
CHESTER CHALLENGER—9. X-ray demonstration.
H. H. HILL—9. Demonstration of pathological specimens.
ROBERT E. FLANNERY—2. Gall-bladder surgery.
LEO P. KOZAKIEWICZ—2. Cæsarean section, indications, contra-indications, demonstrations.

WESLEY MEMORIAL HOSPITAL

Monday

P. B. MAGNUSON—2. Bone surgery.

Tuesday

R. W. MCNEALY—9. Gall-bladder surgery; gastro-intestinal surgery.
C. B. REED—2. Obstetrical clinic, moving picture demonstration of breech delivery, perineorrhaphy and forceps delivery; demonstration of external measurements of intra-uterine child.

Wednesday

PHILIP H. KREUSCHER—9. Joint surgery.
GUY VAN ALSTYNE—9. Osteitis tuberculosa multiplex cystica (Jüngling).

Thursday

G. H. GARDNER and M. T. GOLDSTINE—9. Gynecological clinic, vaginal plastic work.

WOMEN AND CHILDREN'S HOSPITAL

Monday

FRANCES FORD—2. X-ray therapy in malignancies.

Tuesday

BERTHA VAN HOOSEN—9. General surgical operations.
JOSEPHINE MCCOLLUM and BERTHA VAN HOOSEN—10. Demonstrations of morphine and scopolamine anæsthesia in surgery.
O. ZELEZNY—11. Demonstrations of electrocoagulation therapy.

Wednesday

PEARLE STETLER—9. General surgical operations.
WALBURGA KACIN and CLARA OCHS—2. Obstetrical cases, management under scopolamine anæsthesia.
FRANCES FORD—2. X-ray demonstrations.
PEARLE STETLER—2. Surgical diagnosis of appendicitis in children.

Thursday

ALICE CONKLIN—9. General surgery.
Staff—9. Fracture cases.
MARIE ORTMAYER—10. Urological clinic.
AMELIA GIRYOTAS—11. Carcinoma of the pelvis.
ELOISE PARSONS—2. Endocrine therapy in gynecology sterility operations.

Friday

MARY E. WILLIAMS—9. Gynecological operations.
CONSTANCE O'BRITIS—11. General surgical operations.
MARY SPIVACK and FLORENCE HARK—2. Obstetrics.
CHARLES FORD—2. X-ray and diathermy therapy.

AUGUSTANA HOSPITAL

Tuesday

N. M. PERCY and O. E. NADEAU—9. Goiter and genera surgical clinic.

Wednesday

A. T. LUNDGREN and EARL GARSIDE—9. General surgery.
J. W. NUZUM—9. General surgical clinic.
R. J. ODEN—9. General surgical clinic.

Thursday

N. M. PERCY and O. E. NADEAU—9. Goiter and genera surgical clinic.

Friday

A. T. LUNDGREN and EARL GARSIDE—9. General surgery
J. W. NUZUM—9. General surgical clinic.
R. J. ODEN—9. General surgical clinic.

GARFIELD PARK HOSPITAL

Tuesday

JOHN R. HARGER and SAM PLICE—9. Surgery of the stomach; treatment of peptic ulcer.
L. F. MACDIARMID—9. General surgery.

Wednesday

CLAUDE WELDY and JOHN H. PFLOCK—9. Abdominal surgery.

Thursday

J. M. BERGER and FRANK CHAUVET—9. General surgery.

Friday

CLARENCE SAELHOF—9. Dipliasic strains of bacteria from renal lesions; experimental production of lesions with spironenia (spirochæta Pallida).
VINCENT J. O'CONOR—9. Tuberculosis of kidney with review of cases; hydronephrosis, plastic repair of nephropexy.

COLUMBUS HOSPITAL

Tuesday

DANIEL A. ORTH, C. O. LINDSTROM and M. L. HANNAN —9. General surgery.
DANIEL A. ORTH—9. Indications and contra-indications for spinal anæsthesia.
CHANNING BARRETT—9. Gynecological operations.
MINAS JOANNIDES—9. Collapse therapy in pulmonary tuberculosis.
M. J. SEIFERT—10. Surgical treatment of ulcer of the stomach.
MINAS JOANNIDES—2. Surgery of the chest.

Wednesday

CHANNING BARRETT—9. Gynecological clinic.
G. N. BEECHER and M. B. BURNS—9. Emergency surgery in industrial injuries.

Thursday

MINAS JOANNIDES—9. Surgical treatment of abscess of lung.
F. MUELLER and F. MUELLER, JR.—9. Transplantation of bone.
WILLIAM GEHL and T. L. CHENOWETH—9. Urological clinic.
G. N. BEECHER and M. B. BURNS—9. Emergency surgery in industrial injuries.

Friday

DANIEL A. ORTH, C. O. LINDSTROM and M. L. HANNAN —9. General surgery.
M. J. SEIFERT—9. General surgery.

HOSPITAL OF ST. ANTHONY DE PADUA

Monday

THOMAS DWYER—2. Demonstrations in surgical pathology.

Tuesday

LAWRENCE RYAN—9. General surgery.
J. J. SPRAFKA—9. General surgery.
O. J. JIRSA—9. Urology.
L. S. TICHY—2. X-ray demonstration.

Wednesday

R. C. CUPLER—9. General surgery.
JOSEPH ZABOKRTSKY—9. General surgery.
F. W. SLOBE—2. Fracture clinic.
M. A. WEISSKOPF—2. Obstetrics.

Thursday

FRANK J. JIRKA—9. Abdominal operations.
F. B. OLENTINE and R. C. DRURY—9. Thyroid surgery and general surgical clinic.
O. J. JIRSA—9. Urology.
L. S. TICHY—2. X-ray demonstration.

Friday

S. E. DONLON—9. General surgery.
A. A. BONA—9. General surgery.
M. A. WEISSKOPF—9. Obstetrics.

HENROTIN HOSPITAL

Tuesday

CHANNING BARRETT—9. Gynecological operations.
F. LEE STONE—9. Some problems in tubal patency.

Wednesday

JOHN A. GRAHAM—11. Open reduction of fractures.

JACKSON PARK HOSPITAL

Monday

F. L. BARBOUR—2. Dry clinic: Symposium on treatment of pulmonary tuberculosis, surgical and medical.

Tuesday

T. H. KELLEY—9. General surgical clinic.
ARRIE BAMBERGER—10. General surgical clinic.
C. C. CLARK—11. General surgical clinic.
S. B. MACLEOD—2. Fracture clinic.

Wednesday

ARRIE BAMBERGER—9. General surgical clinic.
H. HOYT COX—10. General surgical clinic.
S. W. MARCHMONT-ROBINSON—2. Dry clinic: Hand infections as related to industrial surgery.
H. F. SPIERLING—3. Mortality of appendicitis.

Thursday

ARRIE BAMBERGER—9. General surgical clinic.
T. H. KELLEY—10. General surgical clinic.
G. MARCHMONT-ROBINSON—11. Injection treatment of hæmorrhoids.
E. ALLEN PARSONS—12. Postoperative treatment of ruptured appendix with peritonitis.
R. T. FARLEY—1. Chorio-epithelioma; pseudo Addison's disease; volvulus.
J. J. MOORE—2. Gross surgical pathology.

Friday

A. F. HENNING—9. General surgical clinic.
GEORGE M. LUCAS—10. Gynecological surgery.
C. C. CLARK—11. General surgical clinic.

ILLINOIS CENTRAL HOSPITAL

Tuesday

HUGH M. MACKECHNIE—9. General surgery.
PHILIP H. KREUSCHER—9. Orthopedics.

Wednesday

CHARLES PFEIFFER—9. General surgery.
BEVERIDGE MOORE—9. Orthopedics.

Thursday

S. CLEMENT HOGAN—9. General surgery.
VICTOR LESPINASSE—9. Genito-urinary surgery.

Friday

WILLIAM T. HARSHA—9. General surgery.
JAMES GILL—9. Neurologic surgery.
JOHN J. GILL—9. Obstetrics.
CHESTER GUY and A. H. BAUGHER—9. Pathological conference.

ALBERT MERRITT BILLINGS HOSPITAL

Staff—9, daily. General surgical operations and clinical demonstrations.
WILLIAM ADAMS. Demonstrations in thoracic surgery.
EDMUND ANDREWS. Gall-bladder surgery.
ALEXANDER BRUNSCHWIG. Management of malignant tumors and experimental bone tumors.
E. L. COMPERE, C. H. HATCHER and DR. KEYES. Operations and demonstrations in orthopedic surgery.
LESTER R. DRAGSTEDT. Surgery of the stomach and colon.
C. B. HUGGINS and H. E. HAYMOND. Operations and demonstrations in genito-urinary surgery.
HILGER P. JENKINS. Abdominal surgery.
D. B. PHEMISTER. Bone surgery, operations and demonstrations.

MUNICIPAL TUBERCULOSIS SANITARIUM

Tuesday

CLEMENT L. MARTIN—9. Perianal tuberculosis.
MINAS JOANNIDES—9. Thoracoplasty; phrenic neurectomy.
HENRY C. SWEANY—11. Pathological conference, demonstration of pathological specimens.

Wednesday

DORRIN F. RUDNICK—9. Nephrectomy for tuberculosis of kidney; operative surgery for tuberculosis of the genito-urinary tract.
FRANK FREMMEL and FRANK SMEJKAL—10. Artificial pneumothorax.
FREDERICK TICE, ALLAN J. HRUBY and K. J. HENRICHSEN—2. Diagnostic clinic.

Thursday

JEROME HEAD and RICHARD DAVIDSON—9. Thoracoplasty; pneumolysis; phrenic neurectomy.
K. J. HENRICHSEN—9. Artificial pneumothorax.

Friday

JEROME HEAD and K. J. HENRICHSEN—9. Surgical conference.

OUTPATIENT PNEUMOTHORAX CLINIC

2049 Washington Boulevard

MINAS JOANNIDES, E. L. QUINN, EMIL BUNTA and CLARA JACOBSON—9 and 2, daily. Artificial pneumothorax on ambulatory patients.

ST. BERNARD'S HOSPITAL

Monday

W. G. EPSTEIN—2. General surgery.

Tuesday

W. J. MULHOLLAND—9. General surgery.
H. HOFMANN—9. General surgery.
G. M. CUSHING—2. General surgery.
L. B. DONKLE—2. Genito-urinary surgery.

Wednesday

B. C. CUSHWAY and R. J. MAIER—9. Roentgenological demonstration of anomalies of spine.
J. B. HAEBERLIN—9. General surgery.
W. S. HECTOR—9. General surgery.
J. A. PARKER—2. General surgery.
S. L. GOVERNALE and S. S. MARKIEWICZ—2. Gastro-intestinal operations.

Thursday

J. T. MEYER—9. Thyroid surgery.
F. M. PHIFER—9. Genito-urinary surgery.
W. P. GUNN—9. Gynecological operations.
D. A. VLOEDMAN—2. Gynecological operations.
C. C. GUY—2. Demonstration of unusual specimens.

Friday

A. E. MCCRADIE—9. General surgery.
E. A. RACH and F. J. STUCKER—9. Operative obstetrical problems.

U. S. MARINE HOSPITAL

Wednesday

O. E. NADEAU—9. General surgical clinic.

Friday

O. E. NADEAU—9. General surgical clinic.

EVANSTON HOSPITAL

Monday

JAMES T. CASE—2. X-ray diagnosis and therapy.

Tuesday

WILLIAM R. PARKES—9. Thyroid clinic.
MARCUS H. HOBART—9. General surgical clinic.
DWIGHT F. CLARK—2. Recent advances in the treatment of common fractures.
MARCUS H. HOBART—2. Fracture clinic.

Wednesday

WILLIAM C. DANFORTH—9. Gynecological operations.
CHARLES E. GALLOWAY—9. Gynecological operations.
JEROME R. HEAD—9. Thoracic surgery.
FREDERICK CHRISTOPHER—2. Demonstration of surgica cases.
ROBERT C. LONERGAN—2. Demonstration of orthopedic cases.

Thursday

WILLIAM C. DANFORTH—9. Gynecological operations.
JOHN L. PORTER—9. Orthopedic operations.
WILLIAM C. DANFORTH—2. Obstetrical clinic.
CHARLES E. GALLOWAY—2. Schiller test for the early diagnosis of carcinoma of the cervix.

Friday

FREDERICK CHRISTOPHER—9. General surgical clinic.
FRANCIS D. GUNN—9. Demonstration of surgical pathology.
CHARLES E. POPE—9. Proctological clinic.
J. EVERETT SANNER—2. Urological clinic.

JOHN B. MURPHY HOSPITAL

Monday

JOSEPH KEREKES and R. J. MURPHY—2. Rectal treatment or appendiceal and other pelvic abscesses.

Tuesday

H. E. DAVIS—10. Studies of epiphyseal growth disturbances.

Wednesday

M. J. PURCELL—10. Emergency surgery.
O. H. SCHULZ—10. Observations on treatment of pneumonia.

Thursday

F. O. BOWE—9. Treatment of puerperal infections.
H. R. KENNY and S. J. MARK—10. General surgery.

Friday

A. C. GARVY—10. Diagnosis and treatment of skull fractures.
H. R. KENNY and S. J. MARK—10. Pre-operative treatment in abdominal cases.

EVANGELICAL DEACONESS HOSPITAL

Tuesday

EDWARD HEACOCK—9. General surgery.

Wednesday

PAUL MORF—9. General surgery.

Thursday

A. J. SCHOENBERG—9. Pelvic surgery.

Friday

JOHN PEARL—9. Abdominal surgery; spinal anæsthesia.

CHICAGO MEMORIAL HOSPITAL

Monday

JULIA C. STRAWN and PAUL M. CLIVER—9. Gynecological clinic.

Tuesday

ARTHUR H. CONLEY and FRED M. MILLER—9. Orthopedic and industrial injury clinic.
JAMES E. FITZGERALD—2. Obstetrical clinic.
JOHN P. O'NEIL, J. WILLIAM PARKER and DORRIN F RUDNICK—2. Urological clinic.

Wednesday

CHARLES E. KAHLKE, LAWRENCE L. ISEMAN, ROBERT A. MELENDY and M. L. WEINSTEIN—9. General surgical clinic.
FRANK WRIGHT—9. Colloidal state of the blood in post-operative pneumonia.
GEORGE M. LANDAU—2. Phrenico exeresis and treatment of unilateral tuberculosis.

Thursday

C. R. G. FORRESTER—9. Fracture clinic.
CASPER M. EPSTEIN—9. Oral and plastic surgery.
CHARLES J. DRUECK, SR.—2. Proctology.
HARRY L. MEYERS—2. Gynecological clinic.
WILLIAM L. BROWN—2. Radium clinic.

Friday

PETER S. CLARK, BENNETT R. PARKER and LEO M. ZIMMERMAN—9. General surgical clinic.

LUTHERAN DEACONESS HOSPITAL

Tuesday

GEORGE H. SCHROEDER, JOHN KOUCKY, H. C. WALLACE and G. H. MAMMEN—9. General surgical clinic.

Wednesday

GEORGE H. SCHROEDER, JOHN KOUCKY, H. C. WALLACE, G. H. MAMMEN, R. G. WILLY and G. O. SOLEM—9. Clinical demonstrations.

Thursday

GEORGE H. SCHROEDER, JOHN KOUCKY, H. C. WALLACE and G. H. MAMMEN—9. General surgical clinic.

Friday

GEORGE H. SCHROEDER, JOHN KOUCKY, H. C. WALLACE, G. H. MAMMEN, R. G. WILLY and G. O. SOLEM—9. Clinical demonstrations.

ILLINOIS MASONIC HOSPITAL

Tuesday

E. WHITE—9. Prostatic surgery.
O. C. RITCH—9. Surgery of the kidney.
CLARENCE SAELHOF—9. Tumors of the testicle.

Wednesday

GILBERT FITZPATRICK—9. Obstetrical problems.
CHARLES PARKES and J. R. HARRY—9. Gall-bladder problems.
CARL F. STEINHOFF—9. Medical consideration of thyroid disease.
HUGH MACKECHNIE—9. Surgery of the thyroid.

Thursday

C. K. TIMMONS—9. Surgical considerations of peptic ulcer.
J. F. DAVIS—9. Surgery of the colon.
WALTER FISCHER—9. Orthopedic problems of the feet.

OAK PARK HOSPITAL

Tuesday

JOHN W. TOPE—9. General surgery.
GORDON SWANSON—9. Orthopedic clinic.
ARTHUR CONLEY—9. Management of fractures of the femur.

Wednesday

RALPH SULLIVAN—9. General surgical clinic, treatment of peptic ulcer.
CHARLES FOX—9. Gynecological operations.
CARL UTHOFF—9. Operative cystoscopy.

Thursday

LOUIS RIVER—9. General surgery.
ADOLPH KRAFT—9. General surgery.
CARL UTHOFF—9. Genito-urinary operations.

Friday

JOHN W. TOPE—9. General surgery.
MEREDITH MURRAY—9. Gynecological operations

LITTLE COMPANY OF MARY HOSPITAL

Monday

W. D. STADLE—2. Management of eclamptic patients.

Tuesday

L. L. CHARPIER—9. Management of fractures about the elbow.
J. E. LAIBE—10. Treatment of carcinoma of the bladder.

Wednesday

E. D. HUNTINGTON—9. Gastro-intestinal surgery, complications.

Thursday

L. L. CHARPIER—9. Management of compound fractures.
W. A. MALONE—10. Radium treatment of carcinoma of the cervix.

Friday

A. W. WOODS—9. Gynecological repair operations.
E. D. HUNTINGTON—10. Intestinal obstruction.

AMERICAN HOSPITAL

Tuesday

R. B. MALCOLM—9. Surgical clinic, tumors of the neck.
MAX THOREK and PHILIP THOREK—9. Surgical clinic, carcinoma of the rectum.
W. B. GERHARD—9. General surgical clinic.
FRANK E. SIMPSON—2. Radium treatment of carcinoma of the mouth and tongue.
SOLOMON GREENSPAHN and FREDERICK BOWE—2. Management of placenta prævia.

Wednesday

MAX THOREK and PHILIP THOREK—9. Surgery of the biliary tract.
HORACE E. TURNER and S. GREENSPAHN—9. Casualty surgical clinic.
L. W. BREMERMAN, DAVIS H. PARDOLL and LEON BEILIN—9. Urological clinic.
FRANK E. SIMPSON—2. Radiological clinic, carcinoma of the breast and female genitalia.

Thursday

BENJAMIN GOLDBERG and JOHN F. PICK—9. Indications and technique for surgery of the chest.
FRANK E. SIMPSON—2. Radiological clinic, indications and contra-indications to radium treatment.

RESEARCH AND EDUCATIONAL HOSPITAL

Monday

H. B. THOMAS—1. Orthopedic surgery.

Tuesday

CARL A. HEDBLOM and WILLARD VAN HAZEL—9. Thoracic and general surgery.
L. S. SCHULTZ—9. Oral surgery.

Wednesday

ERIC OLDBERG—9. Neurological surgery.
R. B. MALCOLM—9. Neurological surgery.
H. B. THOMAS—1. Orthopedic surgery.
F. H. FALLS—2. Obstetrical and gynecological clinic.

Thursday

CHARLES B. PUESTOW—9. General surgery.
C. M. MCKENNA—10. Urological clinic; cystoscopies.
WILLARD VAN HAZEL—2. Thoracic surgery.

Friday

CARL A. HEDBLOM and WILLARD VAN HAZEL—9. Thoracic and general surgery.
F. H. FALLS—2. Obstetrical and gynecological clinic.

GRANT HOSPITAL

Tuesday

ANDRE L. STAPLER—9. General surgery.
F. H. FALLS—9. Gynecology.
E. FISCHMANN—9. Vaginal hysterectomy.
A. G. FREY—9. General surgery.
GEORGE ABELIO—9. General surgery.
E. HESS—10. Urology.

Wednesday

E. SEIDLER—9. Midtarsal resection.
A. G. ZIMMERMAN—9. General surgery.

Thursday

B. H. ORNDOFF—9. Electrosurgery.
W. A. STUHR—9. General surgery.
ANDRE L. STAPLER—2. General surgery.

Friday

SYLVAN COOMBS—9. General surgery.
E. W. FISCHMANN—9. Pus tubes.
A. G. ZIMMERMAN—9. General surgery.

POST-GRADUATE HOSPITAL

Monday

B. C. CUSHWAY—2. X-ray diagnosis.

Tuesday

H. SOLOWAY—10. Urological clinic.
EMIL RIES—10. Gynecological operations.
D. SCHLAPIK—2. Intraurethral prostatectomy, moving picture demonstration.

Wednesday

J. C. BOODEL—10. Rectal operations.
LEO ZIMMERMAN—2. Phlebitis.

Thursday

H. L. MEYERS—10. Gynecological operations.
R. A. LIFVENDAHL—11. Gynecological clinic with colposcopic demonstration.

Friday

EMIL RIES—10. Gynecological operations.

CHICAGO LYING-IN HOSPITAL

Staff: FRED L. ADAIR, J. B. DELEE, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, FRANK E. WHITACRE, MANUEL SPIEGEL and H. C. HESSELTINE.

Monday

Staff—2. Obstetrical operations, motion picture demonstration.

Tuesday

Staff—9. Obstetrical and gynecological operations.

Wednesday

Staff—9. Obstetrical and gynecological operations.
Staff—2. Obstetrical clinic, motion picture demonstration.

Thursday

Staff—9. Obstetrical and gynecological operations.
Staff—2. Obstetrical and gynecological dry clinic, motion picture demonstration.

Friday

Staff—9. Obstetrical and gynecological operations.
Staff—2. Obstetrical and gynecological dry clinic, motion picture demonstration.

ST. JOSEPH HOSPITAL

Monday

HUGH MCKENNA—2. Review of traumatic surgery with special reference to fractures.

Tuesday

FRANKLIN B. MCCARTY—9. Surgical anatomy, pathology and surgical treatment of diseases of the gall bladder.
RALPH A. KORDENAT—2. Breast tumors.

Wednesday

HUGH MCKENNA—9. Abdominal surgery, surgery of the large intestine.
WALTER W. VOIGT—9 Puerperal sepsis.
THOMAS J. O'DONOGHUE—2. Obstetrical and gynecological operations.

Thursday

WILLIAM H. G. LOGAN—9. Cleft palate and cleft lip operations.
RALPH C. KORDENAT—2. Gall-bladder surgery.

Friday

L. WADE MARTIN—9. Obstetrical clinic.

WEST SUBURBAN HOSPITAL

Monday

HARRY J. DOOLEY—2. Urological clinic.

Tuesday

WILLIAM J. POTTS—9. The healing of fractures.
OSCAR B. FUNKHOUSER—9. Gall-bladder surgery.
THOMAS I. MOTTER—9. General surgery.
JAMES H. SKILES—9. Gynecological clinic.

Wednesday

JOSEPH L. NORTELL—9. General surgery.
FREDERICK H. FALLS—9. Gynecological clinic.

Thursday

CHARLES E. HUMISTON—9. General surgery.
WARD E. POTTER—9. Thyroid clinic.
LOUIS FAULKNER—9. Interesting obstetrical conditions.
PAUL C. FOX—9. Gynecological clinic.
EUGENE C. PIETTE—9. Pathological demonstration.
HOMER HUMISTON—2. Urological clinic.

HOLY CROSS HOSPITAL

Tuesday

J. Francis Ruzic—9. Gynecological operations; cholecystectomy, high spinal anæsthesia.
E. R. Crowder—9. Some practical considerations regarding the Graham test.
John F. Dybalski—10. Hysterectomy, spinal anæsthesia.
Vincent Torczynski—11. Appendectomy.

Wednesday

Donald Monaco—9. Thyroidectomy; ecture on avertin anæsthesia.
A. R. McCradie—10. Hernia operation.
Paul Lawler—11. Low cervical cæsarean section.

Thursday

Stephen Biezis—9. Gynecological operations.
Michael Strikol—10. Cholecystectomy.
F. F. Fraider—11. Panhysterectomy.

Friday

M. J. Badzmierowski—9. Thyroidectomy; hysterectomy.
Richard Roche—10. Herniorrhaphy.
J. Francis Ruzic—11. Pre- and postoperative therapy.

ST. ANNE'S HOSPITAL

Tuesday

T. E. Meany—10. Orthopedic clinic.
J. L. Knapp—11. General surgery.
J. B. Harney—2. X-ray demonstration.

Wednesday

G. F. Thompson—9. Stomach and intestinal surgery.
J. W. McConnell—10. Gynecology.
J. J. Gearin—11. General surgery.

Thursday

H. J. Dooley—9. Urological clinic.
E. P. Vaughan—9. Gall-bladder surgery.
E. P. Gramer—10. Treatment of head injuries.
J. L. Fleming—11. Pathological obstetrics.

Friday

B. W. Mack—9. General surgery.
Staff—10. Clinical meeting.
D. F. Hayes—11. General surgery.
L. R. Hill—2. Pathological demonstration.

SHRINERS' HOSPITAL

Tuesday

Beveridge Moore and Harold Sofield—9. Orthopedic operations.

Wednesday

Beveridge Moore—2. Demonstration of plaster technique; club foot clinic.

Thursday

Beveridge Moore and Harold Sofield—9. Orthopedic operations.

Friday

Beveridge Moore and Harold Sofield—2. Out-patient clinic.

SOUTH CHICAGO COMMUNITY HOSPITAL

Tuesday

M. E. Finsky—2. Avertin anæsthesia, analysis of 200 cases.
Louis D. Smith—2:30. Tuberculosis of the kidney, presentation of case.

Friday

Joseph J. Lebowitz—2. Fractures and dislocations of the elbow, presentation of cases treated by open operation.
Frank G. Murphy—2:30. Fractures of the upper end of the humerus, presentation of cases.
George G. O'Brien—3. Postoperative evisceration, presentation of case.

FRANCES E. WILLARD HOSPITAL

Tuesday

Allen E. Stewart—9. General surgical clinic.
Frederick Mueller—3. Surgery of bones and joints.

Wednesday

Otis M. Walter—9. General surgical clinic.
Vaughn L. Sheets—10. Diabetic clinic.

Thursday

Joseph F. Jaros—9. Thyroid clinic.

Friday

Victor L. Schrager—9. General surgical clinic.

ALEXIAN BROTHERS HOSPITAL

Tuesday

Malcolm L. Harris, August Zimmerman, Robert Flannery and George L. Apfelbach—9. General surgery.
A. Wochinski and Edward White—9. General surgery.

SURGERY OF THE EYE, EAR, NOSE AND THROAT

COOK COUNTY HOSPITAL

Monday

EARLE B. FOWLER—2. Ophthalmoscopy.
S. PEARLMAN and N. LESHIN—2. Œsophagoscopy and bronchoscopy; surgery of the neck.

Tuesday

THOMAS D. ALLEN—2. External diseases of the eye.
I. MUSKAT—2. Clinical and surgical otolaryngology; plastic surgery of face and nose.

Wednesday

L. T. CURRY—9. Otolaryngology, clinical and surgical cases.
WILLIAM F. MONCREIFF—9. Ophthalmic neurology and ophthalmoscopy.

Thursday

SANFORD R. GIFFORD—9. Ophthalmic surgery.
CHARLES F. YERGER—11. External diseases of the eye.
S. PEARLMAN and N. LESHIN—2. Œsophagoscopy and bronchoscopy; surgery of the neck.

Friday

T. C. GALLOWAY and M. T. LAMPERT—10. Malignancy about the head, diathermy.
THOMAS D. ALLEN—2. Ophthalmic surgery.
I. MUSKAT—2. Clinical and surgical otolaryngology; plastic surgery of face and nose.

ST. LUKE'S HOSPITAL

Monday

EARL VERNON—1. Ophthalmological clinic.

Tuesday

E. FINDLAY and RICHARD GAMBLE—1. Ophthalmological clinic.
J. T. CAMPBELL, JOHN A. CAVANAUGH, HORACE R. LYONS, E. P. NORCROSS and WALTER H. THEOBALD—2. Otolaryngological clinic.

Wednesday

ALVA SOWERS—1. Ophthalmological clinic.
J. T. CAMPBELL, JOHN A. CAVANAUGH, HORACE R. LYONS, E. P. NORCROSS and WALTER H. THEOBALD—2. Otolaryngological clinic.

Thursday

FRANK BRAWLEY and JAMES W. CLARK—1. Ophthalmological clinic.
J. T. CAMPBELL, JOHN A. CAVANAUGH, HORACE R. LYONS, E. P. NORCROSS and WALTER H. THEOBALD—2. Otolaryngological clinic.

Friday

E. FINDLAY and RICHARD GAMBLE—1. Ophthalmological clinic.

ILLINOIS CENTRAL HOSPITAL

Tuesday

HIRAM SMITH—9. Eye clinic.

Wednesday

JAMES H. MCLAUGHLIN—9. Nose and throat surgery.

CHICAGO EYE, EAR, NOSE AND THROAT HOSPITAL

Tuesday

H. B. FULLER—9. Mastoid surgery.
WILLIAM A. HOFFMAN and WILLIAM LINGARD—9. Ear, nose and throat clinic.
WILLIAM A. FISHER—9. Cataract operations.
WILLIAM A. HOFFMAN—9. Eye clinic.
L. SAVITT—10. Removal of tonsils by diathermy.
OSCAR B. NUGENT—11. Eye clinic.
O. M. STEFFENSON—11. Ear, nose and throat clinic.
T. S. KAMMERLING—2. Eye, ear, nose and throat clinic.

Wednesday

O. M. STEFFENSON—9. Tonsil dissection.
WILLIAM A. HOFFMAN and WILLIAM LINGARD—9. Nasal surgery and ear, nose and throat clinic.
OSCAR B. NUGENT—9. Cataract operations.
WILLIAM A. HOFFMAN—9. Eye clinic.
OSCAR B. NUGENT—11. Eye clinic.
O. M. STEFFENSON—11. Ear, nose and throat clinic.
L. SAVITT—11. Ear, nose and throat clinic.
H. B. FULLER—2. Eye, ear, nose and throat clinic.

Thursday

WILLIAM A. FISHER—9. Eye operations.
WILLIAM A. HOFFMAN—9. Eye clinic.
T. S. KAMMERLING—9. Surgery of the nasal accessory sinuses.
WILLIAM A. HOFFMAN and WILLIAM LINGARD—9. Ear, nose and throat clinic.
L. SAVITT—10. Physical measures in otolaryngology.
O. M. STEFFENSON—11. Ear, nose and throat clinic.
L. SAVITT—11. Ear, nose and throat clinic.
OSCAR B. NUGENT—11. Eye clinic.
T. S. KAMMERLING—2. Eye, ear, nose and throat clinic.

Friday

O. M. STEFFENSON—9. Tonsil dissection.
WILLIAM A. HOFFMAN and WILLIAM LINGARD—9. Ear, nose and throat clinic.
OSCAR B. NUGENT—9. Fundus photography and pathology.
WILLIAM A. HOFFMAN—9. Eye clinic.
H. B. FULLER—10. Functional testing.
O. M. STEFFENSON—11. Ear, nose and throat clinic.
OSCAR B. NUGENT—11. Eye clinic.
H. B. FULLER—2. Eye, ear, nose and throat clinic.

MERCY HOSPITAL

Tuesday

GEORGE T. JORDAN—9. Nasal ganglion.
L. G. HOFFMAN—9. Cataract extractions.
C. H. CHRISTOPH—9. Bronchoscopy.

Wednesday

GEORGE MUSGRAVE and ALFRED PAISLEY—9. Frontal sinus operation, local anæsthesia; modified radical mastoid operation with complete removal of flap; presentation of cases.

Thursday

ULYSSES J. GRIM—9. Radical antrum and mastoid.
DENO O'CONNOR and RAY KERWIN—9. Ocular tumors.
CARL SCHAUB—9. Focal infection in iritis.

RESEARCH AND EDUCATIONAL HOSPITAL

Otolaryngological Staff: F. L. Lederer, W. H. Theobald, J. J. Theobald, G. S. Livingston, E. A. Bredlau, N. Fox, S. L. Shapiro, I. G. Spiesman, P. A. Halper, A. C. Kane, A. Coombs, J. Harned, O. Van Alyea, M. Guttman, S. Morwitz, M. Ostrom, B. Lambrakis, E. Hartlett, H. Klawans, L. Fishman, H. Wadsworth, J. Bellows and N. Fabricant.

Ophthalmological Staff: Hallard Beard, M. L. Folk, H. J. Smith, S. Wolf, S. Kaufman, Carl Apple and J. W. Clark.

Monday

Staff—2. Otolaryngological out-patient clinic.

Tuesday

Staff—9. Ophthalmological clinic, operations and demonstrations.
Staff—10. Otolaryngological out-patient clinic.
Staff—2. Otolaryngological clinic, operations and demonstrations.

Wednesday

Staff—9. Eye clinic.
Staff—10. Otolaryngological out-patient clinic.
Staff—2. Otolaryngological out-patient clinic.
Staff—4. Otolaryngological seminar.

Thursday

Staff—9. Otolaryngological operations.
Staff—9. Eye clinic.
Staff—10. Otolaryngological out-patient clinic.
Staff—2. Otolaryngological clinic, operations and demonstrations.
Staff—2. Otolaryngological out-patient clinic.

Friday

Staff—9. Eye clinic, operations and demonstrations.
Staff—10. Otolaryngological out-patient clinic.
Staff—2. Otolaryngological out-patient clinic.

CHICAGO MEMORIAL HOSPITAL

Monday

Richard H. Street and Richard W. Watkins—2. Otolaryngological clinic.

Tuesday

Herman P. Davidson and Glenway W. Nethercut—9. Eye clinic.

Wednesday

Alfred E. Lewy and Irving I. Muskat—2. Otolaryngological clinic.

EVANSTON HOSPITAL

Tuesday

Thomas C. Galloway—9. Otolaryngological clinic.

Thursday

Howard C. Ballenger—9. Otolaryngological clinic.

Friday

Gail R. Soper—2. Lesions of the fundus oculi, lantern slide demonstration.

WASHINGTON BOULEVARD HOSPITAL

Tuesday

L. McBride—2. Nose and throat clinic.

Wednesday

Virgil Westcott—2. Eye clinic.

ILLINOIS EYE AND EAR INFIRMARY

Tuesday

Dwight C. Orcutt—9. Use of flap in cataract work; superior rectus tension suture; plastic.
LeRoy Thompson—9. Industrial ophthalmology.
Carl H. Christoph—10. Bronchoscopy; œsophagoscopy.
M. A. Glatt—1. Radical mastoid and radical frontal operations.
E. R. Crossley—2. Intra- and extra-ocular surgery.
Oscar Cleff—3. Radical mastoid operation.

Wednesday

M. Lebensohn—9. Detachment of retina; cataracts; trephine.
Staff—10. Dry clinic.
Ulysses J. Grim—1. Radical mastoid and radical antrum operations.
Michael Goldenburg—2. Iridotasis operation for glaucoma; cataracts; controlled tenotomy.
John A. Cavanaugh—3. Radical mastoid operation.

Thursday

Herbert Walker—9. Detachment of retina, Larson operation.
C. F. Yerger—10. Radical sinus and radical mastoid operations.
A. Lewy—1. Radical frontal operation.
E. K. Findlay—2. Intra- and extra-ocular surgery.
W. A. Gross—3. Tonsils, diathermy.

MOUNT SINAI HOSPITAL

Monday

Joseph C. Beck, Alfred Lewy, Noah Schoolman, Jacob Lifschutz, S. M. Morwitz and associates—2. Ear, nose and throat operations.

Friday

Joseph C. Beck, Alfred Lewy, Jacob Lifschutz, Noah Schoolman, S. M. Morwitz and associates—9. Ear, nose and throat operations.
James E. Lebensohn—9. Operations for cataract and squint.

Daily, 9 and 2

James E. Lebensohn. Eye changes in hypertensive states.
Alfred Lewy and S. M. Morwitz. Otogenic sepsis.

WEST SUBURBAN HOSPITAL

Monday

Robert H. Good—2. Surgery of the nose, motion picture demonstration.

Tuesday

John J. Theobald—2. Mastoid surgery.

Wednesday

Georgiana Theobald—2. Eye pathological exhibit.

ILLINOIS MASONIC HOSPITAL

Tuesday

M. H. Cottle—10. Some advances in mastoid work.
B. M. Wolin—10. Tonsil surgery in the poor risk cases.
H. E. Taylor—10. Conservative surgery of the nose.

EVANGELICAL HOSPITAL

G. Henry Mundt. Technique and interpretation of hearing tests and technique and interpretation of tests of the static labyrinth.

PRESBYTERIAN HOSPITAL AND RUSH MEDICAL COLLEGE

Monday

D. B. HAYDEN—2. Complications of otitis media without rupture of the tympanic membrane.
E. W. HAGENS—2. Unusual laryngeal and bronchial case.
GEORGE E. SHAMBAUGH, JR. and E. W. HAGENS—2. Operations on the tear sac for dacrocystitis.
MAX JACOBSON—3. Neurological aspects.

Tuesday

ROBERT VON DER HEYDT—3. Slit lamp diagnostic clinic.

Wednesday

VERNON LEECH—3. Glaucoma.

Thursday

BERTHA KLEIN—10. Histopathology of fundus.
T. W. LEWIS—2. Discussion of some difficult problems in the operation for correction of the nasal septum.
L. T. CURRY—2. Demonstration of skiagraphs of the sinuses and mastoids.
R. W. WATKINS—2. Nasal findings in allergic cases.
W. J. JONKER—2. Diathermy and its application to the treatment of nose and throat conditions.

Friday

W. F. MONCPEIFF—10. External diseases of the eye and iridocyclitis.
ELIAS SELINGER—3. Fundus.

MICHAEL REESE HOSPITAL

Monday

H. S. GRADLE—2:30. Eye surgery.

Tuesday

S. J. PEARLMAN—9. Bronchoscopic clinic.
M. L. FOLK—2. Eye surgery.

Wednesday

SAMUEL SALINGER—9. Nasal fractures; plastic of the nose.
M. L. FOLK—2. Eye clinic.
H. S. GRADLE—2:30. Surgical eye clinic.
ROBERT VON DER HEYDT—3. Slit-lamp demonstration.

Thursday

CASPER EPSTEIN—9. Cleft palate and harelip.
S. J. MEYER—2. Eye clinic.

WOMEN AND CHILDREN'S HOSPITAL

Tuesday

ALICE K. HALL—10. Nose and throat clinic.

Wednesday

FRANCES HAINES—10. Nose and throat clinic.

ST. ANNE'S HOSPITAL

Tuesday

B. T. GORDON—9. Nose and throat clinic.

Wednesday

W. K. GRAY—9. Eye and ear clinic.

AUGUSTANA HOSPITAL

Wednesday

ALFRED MURRAY—2. Eye, ear, nose and throat clinic.

ALBERT MERRITT BILLINGS HOSPITAL

Tuesday

E. V. L. BROWN—9. Eye clinic.
J. R. LINDSAY—10:30. Ear, nose and throat clinic.
DEWEY KATZ—2. Eye clinic.

Wednesday

LOUIS BOTHMAN—9. Eye clinic.
T. E. WALSH—10:30. Ear, nose and throat clinic.
JOHN STOUGH—2. Eye clinic.
J. R. LINDSAY and G. H. SCOTT—2. Ear, nose and throat operations.

Thursday

P. C. KRONFELD—9. Eye clinic.
G. H. SCOTT and H. B. PERLMAN—10:30. Ear, nose and throat clinic.
DEWEY KATZ—2. Eye clinic.

Friday

DEWEY KATZ—9. Eye clinic.
J. R. LINDSAY and T. E. WALSH—10:30. Ear, nose and throat clinic.
P. C. KRONFELD—2. Eye clinic.
T. E. WALSH and H. B. PERLMAN—2. Ear, nose and throat operations.

COLUMBUS HOSPITAL

Monday

MICHAEL GOLDENBURG—2. Emergency surgery of the eye.

Wednesday

G. B. LAMBRAKIS—9. Indications for operative treatment in acute mastoiditis.
S. SCIARETTA—9. Otolaryngological clinic.
MICHAEL GOLDENBURG—2. Eye surgery.

Friday

MICHAEL GOLDENBURG—2. Eye surgery.

OAK PARK HOSPITAL

Tuesday

HOWARD RIORDAN—9. Demonstration of new nasopharyngoscope on the cadaver and living.

Thursday

HOWARD RIORDAN—9. Treatment of maxillary sinusitis with the cold quartz lamp; new method of treatment of maxillary polypi by diathermy.

Friday

GEORGIANA THEOBALD—9. Demonstration of eye tumors; ophthalmic surgery.

SOUTH SHORE HOSPITAL

Monday

JOHN W. STANTON—2. Mastoiditis and its complications.

Thursday

JOHN W. STANTON—11. Otolaryngological surgery.

JACKSON PARK HOSPITAL

Tuesday

H. E. L. TIMM—1. Timm's modification of Sluder tonsillectomy.

JOHN B. MURPHY HOSPITAL

Monday

E. F. GARRAGHAN—2. Eye operations.

Tuesday

L. H. WOLF and PAUL WOLF—10. Mastoid surgery.

Friday

GEORGE W. MAHONEY—9. Cataracts.

AMERICAN HOSPITAL

Tuesday

HARRY L. POLLOCK AND ASSOCIATES—2. Ear, nose and throat clinic.

Wednesday

OSCAR KRAFT—2. Ophthalmological clinic.

GRANT HOSPITAL

Wednesday

S. H. SOBOROFF—9. Ear, nose and throat clinic.
GEORGE F. SUKER—9. Eye clinic.
GEORGE DENNIS—9. Eye, ear, nose and throat clinic.

ST. MARY OF NAZARETH HOSPITAL

Tuesday

J. J. KILLEEN—9. Ear, nose and throat clinic.

Thursday

J. J. KILLEEN—9. Ear, nose and throat clinic.

RAVENSWOOD HOSPITAL

Wednesday

A. N. MURRAY—10:30. Malignancies of the eye.

PASSAVANT MEMORIAL HOSPITAL

Friday

J. GORDON WILSON, JOHN DELPH, CARL BOOKWALTER and ELLISON ROSS—9. Ear, nose and throat clinic.
SANFORD GIFFORD, WILLIAM MANN, JR. and RALPH DAVIS—11. Ophthalmology.

CHILDREN'S MEMORIAL HOSPITAL

Wednesday

GEORGE S. LIVINGSTON and staff—9. Otolaryngological clinic.
RICHARD C. GAMBLE and staff—2. Ophthalmological clinic.

FRANCES E. WILLARD HOSPITAL

Thursday

WILLARD D. BRODE—10. Surgery of throat and nose.

LITTLE COMPANY OF MARY HOSPITAL

Wednesday

H. T. NASH—10. Emergency surgery of the eye.

SOUTH CHICAGO COMMUNITY HOSPITAL

Tuesday

GEORGE E. PARK—3. The center of ocular rotation in the horizontal plane.

ST. BERNARD'S HOSPITAL

Friday

PHILIP O'CONNOR—2. Surgery of the eye, dry clinic.